United States
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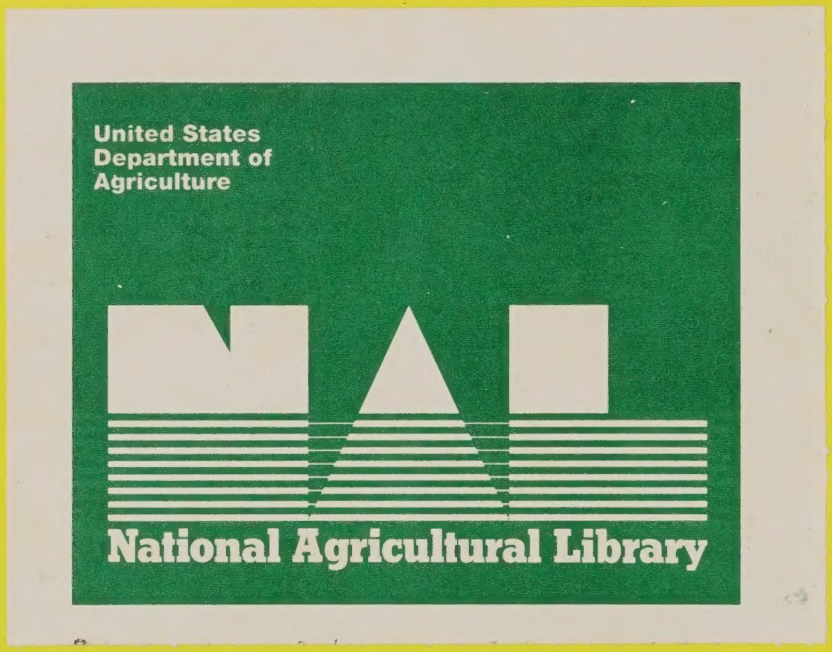
Forest Service

Animal and Plant
Health Inspection
Service

Gypsy Moth Suppression and Eradication Projects

Final Environmental Impact Statement as Supplemented - 1985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Final Environmental Impact Statement as Supplemented 1985
USDA Gypsy Moth Suppression and Eradication Projects
Prepared in Accordance with Section 102(C) of NEPA

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Abstract: This Final Environmental Impact Statement (FEIS) as supplemented in 1985 describes the proposed USDA Forest Service and Animal and Plant Health Inspection Service (APHIS) gypsy moth suppression and eradication projects in cooperation with State and Federal agencies. Four alternatives incuding a "no-action" alternative, are described. The environmental effects of implementing each of the proposed alternatives are discussed. A risk analysis concerning the use of chemical insecticides and their effect on human health is presented. The alternative selected by USDA Forest Service and APHIS is Integrated Pest Management integrating the use of chemical and biological insecticides, and other operational technologies to suppress or eradicate gypsy moth infestations throughout the United States. Annual requests by cooperating State and Federal agencies for USDA Forest Service financial assistance will be considered on an individual basis. Annual decisions concerning USDA Forest Service participation in suppression projects and APHIS participation in eradication projects will be based on the results of site-specific environmental analyses conducted in accordance with NEPA.

Date of transmission to EPA and the public: March 8, 1985

Draft: _____ Final: X

SUMMARY

FINAL ENVIRONMENTAL IMPACT STATEMENT AS SUPPLEMENTED - 1985

USDA GYPSY MOTH SUPPRESSION AND ERADICATION PROJECTS

This Final Environmental Impact Statement contains information and data incorporated from the Draft Supplement which was made available for public review and comment. The Draft Supplement was developed in response to additional comments and new information available since the 1984 FEIS was published.

This document incorporates significant revisions in Appendix F concerning the analysis of human health risks of using acephate, carbaryl, diflubenzuron and trichlorfon insecticides in gypsy moth suppression and eradication projects.

Deletions and additions have been incorporated in the Chemical Insecticides section of the Environmental Consequences Chapter in this document to reflect the revised risk analysis (Appendix F). Minor corrections and revisions have also been made to update and clarify the text of this FEIS.

Purpose of and Need for Action

Since its accidental release in the United States in 1869, gypsy moth has spread throughout New England and areas to the south and west, and is now permanently established in all or parts of 14 States. Most recent additions include the eastern panhandle of West Virginia and northwestern Virginia. The gypsy moth has caused severe tree defoliation on more than 53 million acres since 1924, with 56 percent of that total or 29 million acres occurring during the period 1980-84. Although defoliation has decreased over most of the Northeast during the period 1982-1984, the gypsy moth continues to spread south in Virginia and West Virginia, into western portions of Pennsylvania, and in an earlier established isolated infestation in central Michigan.

An increase in the number of isolated infestations resulting from the artificial movement of gypsy moth life stages to areas outside of the generally infested areas has occurred nationwide.

The gypsy moth has caused dramatic economic impacts in the generally infested areas. In the 1980 report to Congress, USDA estimated losses to homeowners, forest industries, and recreation areas at $272 million. Timber losses have been as high as $72 million in 1981.

Significant economic impacts are predicted outside of the generally infested area if isolated infestations become permanently established. For example, potential losses in California ranging between $446 million and $457 million for the period 1982 to 1999 have been predicted if gypsy moth infestations in the State are not treated.

Major Issues and Concerns

Major issues and concerns were identified during scoping activities. In addition, a 1983 court decision (Oregon Environmental Council vs. Kunzman et al. CA No. 82-3232, DC No. CV82-504) amplified some of these same issues.

Major issues, concerns, and opportunities identified during this entire process were: concern for human health; a need for more public education regarding gypsy moth suppression and regulatory programs; a need for increased public involvement in selection of insecticides and treatment areas; concern for the environmental effects of using insecticides; a need for discussion of alternatives to chemical insecticides; a need for continued Federal/State coordinated gypsy moth suppression and eradication projects; a need for improved and continuous communications between project coordinators regarding safety; and a need to update future National Environmental Policy Act (NEPA) documents with new information on registered insecticides such as label changes, new insecticides, and environmental monitoring.

Alternatives Including Proposed Action

Alternatives considered for USDA gypsy moth suppression and eradication projects on Federal and non-Federal lands are:

(1) No action.

(2) Chemical insecticide treatment.

(3) Biological insecticide treatment.

(4) IPM approach (selected).

The no action alternative would result in no USDA-funded suppression or eradication projects. Technical assistance would be provided by USDA if requested. The no action alternative would not

preclude financing and implementation of suppression and eradication projects by individual States, counties, or private citizens.

The chemical insecticide treatment alternative would result in funding of proposals to use chemical insecticides such as carbaryl, trichlorfon, diflubenzuron, and acephate. These chemical insecticides have successfully achieved the desired project objectives in previous suppression and eradication projects and are registered by the U. S. Environmental Protection Agency (EPA) for application against the gypsy moth. Currently, none of these chemical insecticides are under special review by EPA for suspected health or environmental hazard posed by their registered uses according to generally accepted application practices.

The biological insecticide treatment alternative would result in funding of proposals to use biological insecticides. The biological insecticides registered by the EPA for gypsy moth suppression are formulations of Bacillus thuringiensis kurstaki (B. t.) and the gypsy moth nucleopolyhedrosis virus (NPV).

The IPM approach would result in funding of IPM strategies for gypsy moth suppression and eradication. The components of this strategy include biological and/or chemical insecticide application, parasite and predator management, application of the gypsy moth pheromone, release of sterile or partially sterile gypsy moth life stages, and forest stand manipulation. Currently, only use of the biological and chemical insecticides and the gypsy moth pheromone are considered operationally viable gypsy moth suppression or eradication components.

Affected Environment

Gypsy moth is permanently established in all or portions of Connecticut, Delaware, Maine, Maryland, Massachusetts, Michigan, New Hampshire, New Jersey, New York, Pennsylvania, Rhode Island, Vermont, Virginia, and West Virginia. Natural spread of the insect will likely continue southerly and westerly to adjacent States. Localized isolated infestations presently occur in California, Illinois, Indiana, Michigan, Minnesota, North Carolina, Ohio, Oregon, South Carolina, Tennessee, Virginia, Washington, and Wisconsin. Artificial movement of gypsy moth life stages from the generally infested areas will

continue to cause establishment of isolated infestations in these and other States where suitable host material exists; however, regulation of articles contributing to this movement will reduce such occurrences.

The areas now experiencing and those susceptible to gypsy moth are not homogenous in terms of physical, biological, economic or social attributes. Therefore, specific identification and discussion of the affected environment will be addressed in site-specific environmental analyses for proposed suppression and eradication projects.

Environmental Consequences

The no action alternative would not necessarily eliminate gypsy moth suppression or eradication activities. Nontarget organisms will not be adversely affected. Gypsy moth parasite and predator populations may increase to levels exerting some biological control of localized gypsy moth populations. If the no action alternative were implemented outside of currently regulated areas of the country, Federal and State quarantines would be imposed to limit artificial spread from these areas. If State and other Federal agencies or individuals implement their own suppression or eradication activities, the biological and physical effects would depend on the method used. Untreated infestations on Federal lands could adversely affect suppression and eradication efforts by non-Federal landowners on adjacent land. If no action is taken by State agencies or individuals, there would be no environmental impacts except those caused by the presence of the gypsy moth. Suppression projects undertaken without State coordination may not provide for adequate public involvement and notification of property owners adjacent to those residences conducting suppression or eradication activities. This could result in the application of more insecticide than is necessary to suppress or eradicate gypsy moth populations which could result in unnecessary adverse environmental effects.

Implementation of the chemical insecticide treatment alternative will, in the year of treatment, reduce gypsy moth populations, reduce larval nuisance, protect foliage, and prevent excessive tree mortality. The application of chemical insecticides will, in the year of treatment, lower the risk of artificial spread, reduce populations of nontarget insects, including

some beneficial insects. Diflubenzuron is toxic to some aquatic organisms. Carbaryl is toxic to honeybees, some aquatic insects, and shellfish. Acephate is toxic to some nontarget organisms and honeybees immediately after treatment. Trichlorfon is toxic to flies, including some gypsy moth parasites. These insecticides will be applied in accordance with EPA-approved label directions.

The use of registered chemical insecticides in gypsy moth suppression and eradication projects results in exposures and doses below threshold doses established in laboratory animals. The expected doses should not have an adverse effect on fish, wildlife, livestock, or domestic animals. The risk analysis indicates that all realistic doses and many worst case doses associated with using these insecticides for suppression and eradication projects are below Acceptable Daily Intake (ADI) and are therefore within acceptable margins of safety. The cancer potency of N-nitrosocarbaryl, acephate, 4-chloroaniline, and trichlorfon are discussed. In each case the weighted risk of cancer in a 70 year lifetime is estimated to be about equal to or less than one chance in a million. Some estimated doses and exposures associated with accidental insecticide spills (aircraft and vehicular) could adversely affect human health. These are identified and the probability of the accident occurring is calculated. The registered use of chemical insecticides, as applied to treatment areas during gypsy moth suppression and eradication projects, will have no adverse effect on fish, wildlife, livestock, and domestic animals. Specific risks associated with the possible use of each insecticide are discussed below:

Acephate.--The analysis of human health risks indicates there is little risk of adverse human health effects for either the general public or occupationally exposed individuals as a result of using registered dose rates of acephate in gypsy moth suppression and eradication projects. Possible temporary effects related to cholinesterase inhibition are identified for sensitive populations based upon the worst case assumptions and exposure levels used in the analysis. These are a result of worst case exposure estimates for consumption of food and water that may contain residues of acephate. The only adverse health effects for workers or the general public identified are associated with major insecticide releases such as spills (probability of

occurrence 10^{-4} to 10^{-6}), with a possible exposure to 2 to 3.5 people. Worst case risks of cancer from exposures to acephate range up to 2.4×10^{-6} or slightly more than 2 in one million.

Carbaryl.--For carbaryl, the overall conclusion in the risk analysis is that realistic and most worst case exposures that might result from its use to control gypsy moth are below threshold doses for specific health responses (e.g. birth defects) by a margin of safety established by the EPA. Worst case exposures that include consumption of food and water containing carbaryl residues are below ADI levels, indicating that cholinesterase inhibition could occur. Risk of cancer resulting from exposure to carbaryl is below one in a million (1×10^{-6}). Based on a review of mutagenicity studies, carbaryl poses a low risk for heritable mutations in humans.

Diflubenzuron.--The risk analysis demonstrates that all doses (realistic and worst case) associated with the use of diflubenzuron are equal to or below the established ADI. This suggests that these doses are well within acceptable margins of safety. Finally, on the specific issues of mutagenicity and cancer, diflubenzuron was found to be nonmutagenic even at high dose levels and noncarcinogenic in oncogenicity studies recently reviewed by EPA. Worst case cancer risks associated with exposure to 4-chloroaniline, a metabolite of diflubenzuron, were estimated to be in the order of 1 in 100 million (1×10^{-8}).

Trichlorfon.--Toxicology data reviewed indicate that trichlorfon is a possible human mutagen. Risks of heritable mutations in humans are estimated to be less than 1×10^{-7} or 1 in 10 million. Trichlorfon also causes cholinesterase depressions at low levels of exposure and causes possible teratogenic effects at very high levels. When the estimated exposure levels to the general public are compared to the ADI for trichlorfon, all realistic dose estimates are below the ADI. The realistic doses are thus considered to be below levels for threshold effects (e.g. birth defects) by margins of safety that are greater than 100. The worst case dose estimates that include consuming food or water containing residues of trichlorfon, as well as all worker exposures, are all greater than the ADI. This indicates that some adverse health effects would occur at these dose levels.

The risk of cancer resulting from exposure to trichlorfon was evaluated because of the possible mutagenic and carcinogenic potential of trichlorfon. The lifetime risk to an individual receiving the highest worst case dose is 1.34×10^{-7} or 1 in 10 million.

Implementation of the biological insecticide treatment alternative can be expected to provide foliage protection, population reduction, and have no adverse effect on nontarget organisms, except some lepidopterous larvae. The only biological insecticide currently available for gypsy moth suppression and eradication are formulations of *B. t.* There will not be a direct loss of existing parasite or predator populations in areas treated with *B. t.*; however, some nontarget lepidopterous larvae may be affected. Recently, single applications of *B. t.* have demonstrated effectiveness in suppression projects; however, multiple applications of *B. t.* (2 or more) may be required to achieve project objectives in some areas where gypsy moth populations are extremely high or where eradication is the goal. The biological insecticide derived from the gypsy moth nucleopolyhedrosis virus (NPV), although registered for use, has not demonstrated the consistent efficacy required for operational use. Although the biological insecticides can provide foliage protection, larval mortality does not occur rapidly. Consequently, larval nuisance and tree defoliation are likely to continue for several weeks after application. Implementation of the biological insecticide treatment alternative will not result in irreversible or irretrievable adverse environmental impacts.

Implementation of the IPM alternative would result in the use of biological and/or chemical insecticides, the gypsy moth mating disruption pheromone, and other operationally available suppression and eradication methods in an integrated approach. The biological effects of the IPM alternative will depend on the extent to which the various components are implemented. Risks to the human environment for the various IPM alternatives would be equal to those already identified for the specific biological or chemical treatment. The IPM approach encourages the selection of either biological and chemical insecticides or other control methods commensurate with treatment needs and land management objectives. In terms of foliage protection and population reduction, the IPM alternative may

result in a greater degree of tree defoliation and/or higher gypsy moth population in some areas than may be realized under the chemical insecticide and/or biological alternatives. In some situations, quarantines may be necessary to limit artificial spread from such areas. Economic efficiency for IPM may be less than for the chemical and/or biological insecticide alternatives depending on the extent to which various IPM components are implemented.

Consultation With Others

In accordance with NEPA regulations, the Draft Supplement was sent for public review and comment to a variety of agencies, organizations, and individuals. Written comments were received from the following agencies and individuals:

Maryland Department of State Planning
North Carolina State Clearing House
Oregon State Clearing House
Ohio State Clearing House
Tennessee Historical Commission
Iowa State Clearing House
Rhode Island Office of State Planning
Missouri State Clearing House
Washington Department of Ecology
Oregon State Clearing House
National Network to Prevent Birth Defects
Northwest Coalition for Alternatives to Pesticides
Pesticides Hazards Clearing House
Oregonians for Food and Shelter
North Carolina State Clearing House
South Carolina Office of the Governor
National Coalition Against the Misuse of Pesticides
New Jersey Department of the Public Advocate
Virginia Council on the Environment
Kenneth and Janet Nolley
Glen and Elaine Olsen
New Jersey Coalition for Alternatives to Pesticides
Pennsylvania Intergovernmental Council
The Resources Agency of California
United States Department of Health and Human Services
Ohio State Clearing House
Chevron Chemical Company
United States Department of Interior
United States Environmental Protection Agency
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INTRODUCTION

This Final Environmental Impact Statement (FEIS) as Supplemented 1985 describes four alternatives developed by the USDA Forest Service (FS) and Animal and Plant Health Inspection Service (APHIS) for suppressing or eradicating gypsy moth infestations on Federal and non-Federal land in cooperation with State and Federal agencies. The alternatives are evaluated and the preferred alternative is identified. The alternative implemented will guide USDA participation in gypsy moth suppression and eradication projects. When new insecticides, technology, or application methods are developed, or when environmental analyses identify unreasonable adverse effects to human health or the environment, appropriate action will be taken.

Decisions concerning USDA Forest Service and APHIS participation in suppression and eradication activities will be based on the results of site-specific environmental analyses conducted in accordance with the National Environmental Policy Act (NEPA).

Table 1. Summary of gypsy moth-caused defoliation (acres) by State, 1924-1984.

Year	ME	NH	VT	MA	RI	CT	NY	PA	NJ	DE	MD	MI	VA	Total
							acres							
1924	71	591	0	163	0	0	0	0	0	0	0	0	0	825
1925	0	239	0	48,321	0	0	0	0	0	0	0	0	0	48,560
1926	1	960	5	78,193	1,663	0	0	0	0	0	0	0	0	80,822
1927	4,985	3,923	2	131,880	126	4	0	0	0	0	0	0	0	140,920
1928	5,575	119,757	3	137,121	58	0	0	0	0	0	0	0	0	262,514
1929	15,187	440,845	0	95,078	23	0	0	0	0	0	0	0	0	551,133
1930	55,174	205,125	0	27,856	66	5	0	0	0	0	0	0	0	288,226
1931	20,938	96,690	277	86,694	114	8	0	0	0	0	0	0	0	204,721
1932	42,298	43,287	1	200,387	376	46	0	0	0	0	0	0	0	286,395
1933	19,718	216,669	2	157,003	4,292	46	0	0	0	0	0	0	0	397,730
1934	60,403	285,880	25	128,237	17,750	66	0	0	0	0	0	0	0	492,361
1935	92,630	330,195	106	106,097	10,908	833	0	0	0	0	0	0	0	540,769
1936	80,944	192,114	0	152,469	3,095	0	0	0	0	0	0	0	0	428,622
1937	140,026	72,973	81	393,613	2,063	4	0	0	0	0	0	0	0	608,760
1938	120,432	34,122	416	154,348	3,297	1,339	0	0	0	0	0	0	0	313,954
1939	202,193	136,772	5,311	143,292	848	4,224	0	0	0	0	0	0	0	492,640
1940	204,041	152,797	3,160	125,586	52	0	0	0	0	0	0	0	0	485,636
1941	122,386	80,579	980	263,369	707	0	0	0	0	0	0	0	0	468,021
1942	850	6,963	49	36,715	0	0	0	0	0	0	0	0	0	44,577
1943	10	290	0	34,481	64	0	0	0	0	0	0	0	0	34,845
1944	21,221	2,346	210	225,637	640	14	75	6	0	0	0	0	0	250,149
1945	210,881	58,517	93,950	456,832	1,280	16	0	11	0	0	0	0	0	821,487
1946	203,813	183,943	15,900	217,132	1,645	486	0	0	0	0	0	0	0	622,919
1947	0	166	0	7,256	0	0	0	0	0	0	0	0	0	7,422
1948	60	21	0	32,386	0	0	0	0	0	0	0	0	0	32,467
1949	0	8	0	78,665	0	0	0	0	0	0	0	0	0	78,673
1950	2	12	0	4,979	0	375	0	0	0	0	0	0	0	5,368
1951	8,195	2,478	1,108	3,185	0	5,673	675	0	0	0	0	0	0	21,314
1952	82,715	94,975	26,985	82,372	0	6,005	0	0	0	0	0	0	0	293,052
1953	174,999	209,335	120,787	917,996	0	56,215	7,745	0	0	0	0	0	0	1,487,077
1954	170,485	154,015	24,650	118,095	0	13,848	10,355	0	0	0	0	0	0	491,448
1955	10,810	14,975	8,875	0	0	6,842	10,559	0	0	0	0	0	0	52,061
1956	7,285	9,305	12,635	3,830	0	3,458	6,645	0	0	0	0	0	0	43,158
1957	120	0	495	16	0	4,909	858	60	0	0	0	0	0	6,458
1958	0	0	0	8	0	117	0	0	0	0	0	0	0	125
1959	1,000	4,000	1,500	382	0	5,980	1,605	0	0	0	0	0	0	14,467
1960	6,350	4,600	6,132	150	0	15,000	16,490	0	0	0	0	0	0	48,722
1961	21,340	621	11,834	3,000	0	0	30,685	0	0	0	0	0	0	67,480
1962	3,998	3,390	6,292	150,000	0	83,290	61,342	0	0	0	0	0	0	308,312
1963	1,970	8,345	12,020	87,847	0	40,140	22,600	0	0	0	0	0	0	172,922
1964	0	14,509	23,523	20,787	375	98,552	97,237	0	0	0	0	0	0	254,983
1965	190	8,451	2,903	17,232	50	86,009	148,366	0	0	0	0	0	0	263,201
1966	30	20	650	500	110	15,895	34,655	0	5	0	0	0	0	51,865
1967	825	561	2	909	150	2,731	46,160	0	1,035	0	0	0	0	52,373
1968	777	5,830	0	3,925	565	16,416	47,525	60	5,025	0	0	0	0	80,123
1969	1,450	17,160	0	6,060	313	56,881	121,610	830	51,525	0	0	0	0	255,829
1970	1,080	38,525	0	6,835	1,082	368,706	416,270	10,500	129,835	0	0	0	0	972,833
1971	820	3,250	790	18,787	8,525	655,107	479,150	598,200	180,595	0	0	0	0	1,945,224
1972	40	200	4,215	20,480	22,510	513,880	177,605	404,060	226,140	0	0	0	0	1,369,130
1973	490	30	200	43,970	35,925	333,215	248,441	856,710	254,865	0	0	0	0	1,773,846
1974	860	0	0	76,903	2,120	120,980	42,350	479,590	28,102	0	0	0	0	750,905
1975	110	0	15	17,895	435	63,411	9,275	317,880	55,430	0	0	0	0	464,451
1976	0	0	1,750	29,820	7,540	9,809	26,583	732,310	57,630	0	0	0	0	865,442
1977	2,010	320	33,435	133,081	125	0	91,313	1,296,550	39,185	0	0	0	0	1,596,019
1978	4,120	725	29,756	63,042	0	3,835	500,046	452,892	204,830	0	0	0	0	1,259,246
1979	23,180	5,980	15,411	226,260	655	7,486	162,275	8,552	193,700	10	0	100	0	643,609
1980	221,220	183,999	75,094	907,075	43,830	272,213	2,449,475	440,500	411,975	0	3	5	0	5,005,389
1981	655,841	1,947,236	48,979	2,826,095	272,556	1,482,216	2,303,915	2,527,753	798,790	500	8,826	18	0	12,872,725
1982	574,537	878,273	9,864	1,383,265	658,000	803,802	825,629	2,351,317	675,985	1,265	9,162	92	0	8,171,191
1983	16,285	560	0	148,133	53,880	153,239	290,843	1,360,824	340,285	2,992	15,870	457	0	2,383,368
1984	41,824	0	0	185,520	164,600	544	33,678	444,900	98,695	14,203	41,824	6,425	374	992,655
Total	3,658,795	6,277,452	600,378	11,027,245	1,322,413	5,313,870	8,722,035	12,283,505	3,753,632	18,970	75,685	7,097	374	53,021,519

PURPOSE AND NEED FOR ACTION

BACKGROUND

The Situation

The gypsy moth, Lymantria dispar L., is native to many areas of Europe, Asia, and Africa. This insect was accidentally released in the United States in 1869 in Massachusetts. In 1924, only 3 States reported the presence of gypsy moth defoliation; however, by 1984 gypsy moth defoliation had been reported in 13 States. Of concern is the total cumulative defoliation recorded since 1924 (53,021,519 acres), particularly the recent rapid increases as depicted in the following tabulation:

Period	Total Defoliation (acres)	Percent of Cumulative Total
1924-69	11,955,486	23
1970-79	11,640,705	22
1980-84	29,425,328	56

A summary of gypsy moth-caused defoliation by State from 1924 to 1984 is presented in Table 1.

The current gypsy moth outbreak began in 1980 when more than 5 million acres were defoliated in the Northeastern States and Michigan. This represented a record in defoliated acres, more than 2.5 times the previous high total observed in 1971. Gypsy moth activity increased dramatically in 1981 and caused tree defoliation on more than 12.8 million acres. New State defoliation records were reported that year in Connecticut, Delaware, Maine, Maryland, Massachusetts, New Jersey, New Hampshire, Pennsylvania, and Rhode Island. Insect activity declined slightly in 1982, causing defoliation on 8.1 million acres. Although defoliation levels decreased over most of the Northeast that year, insect activity was particularly brisk along the leading edge of the infestation as the gypsy moth continued its spread in Maryland, Delaware, southern and western portions of Pennsylvania, and in an earlier established infestation in central Michigan.

An apparent natural collapse of gypsy moth populations began throughout much of the Northeast in 1983 as defoliation levels dipped to slightly more than 2.3 million acres (the 4th highest level of defoliation recorded). The insect, however, accelerated its activity in Maryland, Delaware, and central Michigan with all three States reporting record levels of defoliation.

History of Gypsy Moth Control 1/

Gypsy moth control activities have been cyclical due to the periodic increases and decreases in the gypsy moth population. Control activities have traditionally involved Federal, State, county, and local government as well as private citizens.

Inconspicuous during the first 20 years after its introduction, gypsy moth populations exploded in 1889, threatening to overrun Medford, Massachusetts. Unable to deal with the situation, local officials appealed for State assistance. The Commonwealth responded quickly and appropriated funds for control activities and for a permanent commission to carry out the work. Soon thereafter, Gypsy Moth Commissioners met with mayors of affected towns, scientists, and officials from the USDA Division of Entomology to seek advice on long-range goals and tactics. Largely at USDA urging, a policy of eradication was implemented.

The first recorded use of a chemical to control gypsy moth occurred as part of the Massachusetts eradication effort. The material was an arsenical, Paris green (Forbush and Fernald 1896). The treatment of infested trees and other foliage with Paris green was supplemented by applications of creosote to egg masses, burning of infested trees, shrubbery, and clusters of caterpillars, and banding of trees with burlap and sticky materials to either trap the larvae or prevent their ascent of the trees.

The technology of insecticide application lagged far behind scientific understanding of the problem; machines designed for spraying orchards bogged down in the rough, hilly forest terrain. By the end of the 1891 field season it was evident that Paris green could not be used to eradicate the gypsy moth. Besides poor efficacy, Paris green was often phytotoxic and was easily washed off foliage (Kirkland 1905). The inadequacies of Paris green also generated adverse public reaction.

Once again the Gypsy Moth Commission was forced to turn to the expensive and time consuming methods of gathering egg masses and using traps and sticky bands to stop the larvae. Experimental work on new insecticides continued and, while an effective eradication material was not available in 1892, bromine and chlorine were found to be useful in destroying egg masses in hollow trees. In 1893, a new compound, lead arsenate, proved effective in the field (Forbush and Fernald 1896). For the next 50 years, this material was the standard insecticide used in the Northeast for gypsy moth control.

1/ Major sources of this section: USDA 1981a; Dunlap 1980.

In 1900, funds were not appropriated by the Massachusetts legislature for the Gypsy Moth Commission and the first gypsy moth campaign ended. In the succeeding years, gypsy moth populations increased such that during the summer of 1905, insect conditions in Medford, Massachusetts, and surrounding towns were similar to those in 1889.

By 1905, the gypsy moth had spread to other States in New England, making an eradication policy impossible with the insecticides and application methodology available at the time. At this point, the USDA Bureau of Entomology became involved in gypsy moth control activities.

In this second campaign, Federal and State officials looked toward biological control, that is, the use of parasites and predators to keep gypsy moth populations below damaging levels. The USDA funded programs to import and establish natural enemies of gypsy moth from Europe, and States enthusiastically supported the European parasite expeditions. Expectations ran so high that some officials predicted that the biological program would control gypsy moth within 2 to 3 years.

By 1908, however, scientists were having doubts about biological control as an immediate and economical method to control gypsy moth populations. The establishment of gypsy moth parasites and predators was proving more difficult and expensive than had been anticipated. Little was known about the biology and habits of these insects and most parasites did not survive in the new environment. Even though some parasites were parasitizing gypsy moth, the effect was not noticeable.

Biological control eventually did prove somewhat successful. By the 1920's, parasites and predators were having an effect on gypsy moth populations--but not to the extent of providing relief from defoliation and the nuisance of larvae as originally expected. Biological control work slowed after 1911 and continued at a reduced rate until World War I and for a few years beyond.

After 1911, State and Federal agencies fell back to a selective approach, treating areas such as roadsides, parks and town-areas where gypsy moth damage would be visible. In 1922, the gypsy moth reached New York and a barrier zone along the Hudson River was established to confine the insect to New England. A strenuous attempt was made to eradicate isolated infestations outside the barrier zone.

During this period the most important control techniques involved the use of insecticides, mainly lead arsenate. The popularity of insecticides was due to many factors. First was the public's desire for a immediate solution to high,

damaging gypsy moth population levels. Second, insecticides could be used without much advance planning and they had immediate, visible results. Finally, they could be used by individuals or towns without a need to coordinate activities. Chemical insecticides and improved application equipment made roadside and urban spraying economical and practical.

The barrier zone policy continued until 1938 when a hurricane apparently facilitated the spread of the gypsy moth. In the early 1940's, eradication of the gypsy moth in New England was all but abandoned, primarily because lead arsenate and existing equipment were inadequate for large-scale control efforts.

The New York barrier zone was established in part to slow the spread of gypsy moth until new control techniques and methodologies could be developed. In 1939, the insecticidal properties of a synthetic organic chemical, dichloro-diphenyl-trichloroethane (DDT), were discovered.

DDT acted as both a contact and a stomach poison; larvae not killed by contact soon succumbed following ingestion of treated foliage. Less than a pound of DDT per acre killed almost all of the larvae. Soon after DDT was available for general use, USDA officials considered ideas of complete gypsy moth eradication--the first revival of that idea since the turn of the century. By 1956 Federal and State officials had formulated an eradication plan. The first phase involved aerial application of DDT to eliminate outlying gypsy moth infestations in New York, Pennsylvania, and Michigan. The second phase would involve treatment of gypsy moth infestations in New England. At its peak use in 1957, more than 2 million acres of forest and forested communities were treated with DDT. During the period of its use, DDT was applied to more than 12 million acres of forest in 9 Northeastern States and Michigan for gypsy moth control (EPA 1975).

In the late 1950's and early 1960's, a growing public concern developed over the use of DDT. The material was being described as a "dangerous substance which killed beneficial insects, upset the natural ecological balance and collected in the food chain, thus posing a hazard to man, and other forms of advanced aquatic and avian life" (EPA 1975). Beginning in 1958, DDT was phased out of USDA cooperative gypsy moth suppression projects. In 1972, the EPA cancelled most uses of DDT.

Since the 1950's, there has been an increase in the research and development of new insecticides. In 1958, a new material, carbaryl, under the trade name Sevin®, was introduced to replace DDT as the primary agent to control the gypsy moth. Although carbaryl has a much shorter

half-life and is generally considered much safer than DDT, the material in certain formulations is highly toxic to honeybees. During the period from 1962 to 1977, almost 2 million pounds of this material were used by Federal and State agencies against the gypsy moth in the Northeastern United States.

In the late 1960's, an organophosphate, trichlorfon, registered under the name of Dylox®, proved efficacious against the gypsy moth. By the early 1970's, 2 formulations of Dylox had been used in operational gypsy moth programs. During the 1970's, another organophosphate, acephate (Orthene®) and an insect growth regulator, diflubenzuron (Dimilin®), were registered. Additional chemicals such as malathion, methoxychlor, and phosmet are registered for gypsy moth control, but they are not generally used in Federal/State suppression or eradication projects.

Insecticide research and development in recent years has not been limited to chemicals. During the 1950's, USDA began development on a bacterium that affects many lepidopterous species. This bacterium, Bacillus thuringiensis (B. t.) is currently registered and available under a variety of trade names. During the 1960's, the USDA Forest Service began investigations of a nucleopolyhedrosis virus (NPV) that causes a wilt disease primarily in heavy gypsy moth populations. This virus product was refined in the 1970's and registered in 1978. It is currently undergoing field testing and is not considered ready for operational use at this time. Also in the 1970's, the USDA successfully isolated and synthesized the sex attractant emitted by female gypsy moths. This material, called disparlure, has been used almost exclusively as a detection tool for locating isolated gypsy moth infestations outside of the Northeast. Development and evaluation of the material to confuse male moths and disrupt mating is currently being conducted by Federal agencies and private industry.

Gypsy moth suppression activities have evolved from State-administered projects like those in Massachusetts in the 1890's, to current Federal/State coordinated projects. The failure of earlier policies to check the natural spread of gypsy moth caused pest managers and private citizens during the 1950's to no longer attempt eradication of the insect in the Northeast. Although gypsy moth eradication is still the goal in isolated infestations, strategies are tailored to fit the particular situation in an Integrated Pest Management (IPM) approach.

An IPM approach is possible today because of the different treatments and methodologies now available or soon to be operational. These include more refined survey methods and ability to predict population buildups and subsequent impacts. The existence of several chemical and biological

insecticides allows pest managers flexibility in selecting tactics that are most effective and that have a minimal impact on the environment. An IPM strategy also provides for more public involvement in the selection of treatment areas and tactics.

Major Issues and Concerns

The general buildup of gypsy moth populations has focused public attention on efforts to suppress infestations and regulate spread. Major concerns of State agencies responsible for gypsy moth management and other Federal landowners are the impacts of establishment of isolated infestations, larval nuisance, tree defoliation, and tree mortality.

In developing this FEIS for gypsy moth suppression and eradication, the USDA Forest Service and APHIS sent letters to Federal, State and local agencies, private industry, environmental and related private organizations, and interested individuals (Appendix A). These groups and individuals were asked to identify relevant issues and concerns. In addition, a 1983 court decision (Oregon Environmental Council vs. Kunzman et al, CA No. 82-3232, DC No. CV 82-504) amplified some of these same issues. The major issues and concerns identified through the public scoping process and the court cases, beginning with those most frequently mentioned, are:

(1) Human health. Concerns were expressed regarding the aerial application of chemical and biological insecticides to communities and adjacent populated areas in relation to direct and indirect contamination of drinking water, wells, watersheds, and garden crops. Also expressed were the potential health risks from direct and indirect human (including children and sensitive persons) exposure to insecticides, specifically with regard to the supposedly carcinogenic effects of nitrosocarbaryl, and allergies to chemical insecticides. Further concerns were expressed relating to inadequacies of the EPA registration process. Concerns expressed in support for suppression of the insect were related to potential allergic reactions from contact with larval hairs, larval excrement, and moth wing scales. Effects of insecticide applications on forest lands as opposed to lands without tree cover needs to be discussed.

(2) Public education. Participants expressed a need for increased education in the form of publications, newspaper articles, and films that present the gypsy moth problem in an unbiased manner; additional publications on homeowner self-help techniques and the current status of new techniques; increased use of biological insecticide and parasites; and case studies on the long-term effects of gypsy moth-caused

defoliation. In addition, respondents felt that a description of treatment areas and discussion of treatment techniques used in USDA suppression and eradication projects was necessary.

(3) Public involvement and notification. Respondents indicated a need for local involvement in the determination of project criteria and procedures based on State and local meetings and guidance; the need for improved and continuous communications between State, local, and community coordinators and the public regarding areas planned for treatment, treatment dates, cancellation of treatments, and rescheduled dates; and an explanation of plans to ensure public safety. A need also was expressed for the identification of officials responsible for administering suppression or eradication projects and those available to the public to provide other project information.

(4) Environmental effects. Concern was expressed regarding the need to use short residual insecticides and on the effect of insecticides on gypsy moth parasites, predators, honey bees, aquatic insects, and wildlife.

(5) Alternatives to chemical insecticides (New Technologies). Respondents expressed the desire for a discussion of alternatives to chemical insecticides such as the increased use of biological insecticides, homeowner self-help techniques, parasites and predators, and of the effectiveness and long-term benefits of these alternatives.

(6) Availability of past and current Environmental Impact Statements. Some people did not know where to obtain copies of past and current Environmental Impact Statements. A mechanism needs to be developed whereby documents can readily be obtained.

(7) Label interpretation. Participants expressed a need to update Environmental Impact Statements with information on registered insecticides, label changes, new insecticides, monitoring, and human health studies.

(8) Project administration. Requests were made for a more coordinated approach between the USDA Forest Service, APHIS, and cooperating State agencies, a new funding arrangement for cooperative suppression favoring increased application of biologicals over the chemical insecticides, and increased emphasis on IPM.

(9) Federal involvement. Most participants favored State and Federal involvement in cooperative suppression and eradication projects to provide for coordinated projects using registered insecticides applied under optimum weather conditions with proper application timing.

These issues and concerns were used to guide the environmental analysis documented in this FEIS. Issues and concerns dealing with individual projects and techniques to be used will guide site-specific environmental analyses, and be conducted in accordance with NEPA.

Economic Considerations

Economic losses resulting from gypsy moth infestations in the Northeast have been dramatic. USDA Forest Service and APHIS reported to Congress that losses to homeowners, forest product industries, and recreation areas were $272 million in 1980 (USDA 1981b). Timber losses alone in 1981 were estimated at $72 million (USDA 1982a,b). In New Jersey and Pennsylvania, timber loss has been particularly severe in the last decade.

In one study on Stokes State Forest in northwestern New Jersey, the 1979 and 1980 gypsy moth infestations killed more than 15.5 million board feet and 145,000 cords of timber. This in effect reduced the oak growing stock on the forest by 50 percent. Economic losses on this forest alone were estimated to be more than $3 million in a State where the estimated stumpage value of forest products harvested annually is $8.6 million (NJ DEP 1982).

In another study, the Pennsylvania Department of Environmental Resources estimated timber losses in State woodlands resulting from gypsy moth infestations during the 1970's. Based on surveys of 2.2 million acres, it was estimated that more than 545 million board feet of sawtimber and 462 million cubic feet of pulpwood were lost. This represents an average stand loss of 20 percent, valued at more than $36 million (PA DER 1980).

Significant gypsy moth economic impacts are predicted outside of the generally infested area if isolated infestations are not eradicated. In a study prepared for the California Department of Food and Agriculture, Galt (1983) estimates that urban, agriculture, and forestry losses could range between $446 million and $457 million between 1982 and 1999 if isolated gypsy moth infestations are allowed to spread.

USDA PARTICIPATION

Statutory Authority

Laws applicable to the USDA Forest Service and APHIS that govern participation in suppression and eradication projects are:

(1) The Cooperative Forestry Assistance Act of 1978 (P. L. 95-313), which incorporated provisions of the Forest Pest Control Act of 1947 (now repealed), provides authority for Federal/ State cooperation in forest insect and disease management. The law recognizes that the Nation's capacity to produce renewable forest resources is significantly dependent on non-Federal forest lands. Therefore, the Secretary of Agriculture is authorized to assist in the control of forest insects and diseases on non-Federal forest lands of all ownerships to (a) enhance the growth and maintenance of trees and forests and (b) promote the stability of forest-related industries, and employment associated therewith, through protection of forest resources.

(2) The Plant Quarantine Act of 1912 as amended (7 USC 151-165, and 167); the Federal Plant Protection Act of 1957 (7 USC 150aa-150jj); and the cooperation with States in Administration and Enforcement of Certain Federal Laws approved September 2, 1963 (7 USC 450). These statutes authorize among other things the development of APHIS activities for the regulation of the artificial spread of the gypsy moth from the quarantined area, and the eradication of isolated gypsy moth infestations outside this area.

(3) The National Environmental Policy Act of 1969 (P. L. 91-190 42 USC 4321 et seq) requires detailed environmental analysis of proposed major Federal actions that may affect the quality of the human environment. Generally, the courts have regarded those State actions which involve potential environmental consequences, and for which purpose Federal funds are granted, as Federal actions (Atherton 1977).

(4) The Federal Insecticide, Fungicide, and Rodenticide Act of 1947 (7 USC 136) as amended requires that insecticides used in suppression and eradication projects be registered by the EPA.

Agency Goals

The following USDA goals are considered in the evaluation of gypsy moth suppression and eradication projects:

(1) A principal USDA goal is to assure an adequate supply of high-quality food and fiber and a quality environment for the American people. The USDA gives special emphasis to the development and use of efficient and environmentally acceptable integrated pest management systems. All methods, including the use of chemical pesticides, are considered in integrated pest management projects.

(2) Forest Service policy is to protect and preserve the forest resources of the Nation against destructive forest insects and diseases. Pest outbreaks will be prevented or suppressed by methods that will restore, maintain, and enhance the quality of the environment. These objectives are attained on non-Federal lands through cooperation with State foresters or equivalent State officials. Pests are suppressed directly on National Forest System lands and in cooperation with responsible officials on other Federal lands. Projects approved for cooperative financing must meet Forest Service standards for environmental, biological, and economic acceptability and must meet Forest Service Federal role criteria (FSM 3430). Approval is based on the results of an environmental analysis conducted in accordance with NEPA regulations.

(3) The goal of the APHIS/State cooperative regulatory program is twofold: to retard or prevent the artificial, long-distance spread of the gypsy moth and to eradicate isolated infestations when detected. This is accomplished by enforcement of regulations on the movement of articles that contribute to this artificial spread. The major articles regulated are nursery plants, logs and pulpwood, outdoor household articles, firewood, and mobile homes and recreational vehicles. APHIS also is charged with detection and eradication of infestations subsequently established as a result of the artificial movement of gypsy moth life stages into unregulated areas. Only APHIS eradication projects are fully addressed in this document. Cooperation with State agencies in eradication projects is based on the availability of Federal funds, a mutually agreed upon plan of work, and the results of site-specific environmental analyses conducted in accordance with NEPA. Gypsy moth surveys provide information about pest distribution that serve to guide both regulatory and eradication activities.

As a general rule, Federal participation in eradication projects will only occur when gypsy moth populations are identified that are: 1) geographically removed from areas known to be generally infested, 2) the result of artificial spread as opposed to natural spread, and 3) well defined by delimiting traps. An exception to item 3 would be where reproducing populations are found as evidenced by egg masses without adequate delimiting trapping. Precautionary treatments may be prudent prior to delimit trappling in such situations.

The present USDA Forest Service suppression goals should not be confused with eradication policies of earlier years (pre 1960's) in the Northeast. No attempt is being made to treat all of the areas infested. In fact, Federal/State

suppression projects usually treat less than 10 percent of the areas infested in any given year. Parasites, predators, and natural mortality factors are being relied on to exert biological pressures on the majority of gypsy moth infestations. Treatment of localized high-value and high-use areas in suppression projects is intended to meet short-term objectives identified by cooperating State and Federal agencies. Regulatory activities (quarantines, inspections, and treatments) by APHIS lower the risk of artificial spread of the gypsy moth.

ALTERNATIVES INCLUDING THE PROPOSED ACTION

ALTERNATIVES CONSIDERED

The alternatives presented in this FEIS meet the State and Federal suppression and eradication objectives, address issues and concerns raised through scoping activities, and adhere to USDA guidelines governing Forest Service and APHIS participation in suppression and eradication projects.

The four alternatives considered and evaluated are:

(1) No action.

(2) Chemical insecticide treatment.

(3) Biological insecticide treatment.

(4) Integrated Pest Management (IPM) (selected).

Three additional alternatives were considered but eliminated from detailed study because all are still undergoing field testing and development, and as individual alternatives, none has demonstrated the effectiveness necessary for meeting gypsy moth suppression and eradication objectives. These alternatives are:

(1) Parasite and predator management.

(2) Release of sterile or partially sterile gypsy moth life stages.

(3) Intensive forest stand manipulation (silvicultural control).

Although not fully developed, these alternatives are presented and discussed as possible components of the IPM alternative (#4).

COMPARISON OF ALTERNATIVES

No Action

The no action alternative in this document means that no USDA-funded suppression or eradication projects will be conducted on State, private, or Federal lands. However, technical assistance would still be available. Isolated infestations would be subject to regulatory action imposed by APHIS or State regulatory agencies in the form of quarantines, inspections, and some type of treatment of infested materials shipped from the quarantine areas.

Selection of this alternative, however, would not preclude some type of action taken by State, municipal, or private individuals to suppress or eradicate gypsy moth populations.

Therefore, some suppression of outbreak populations may occur within the infested northeastern States. In addition, communities or towns may elect to finance their own suppression as may individual land owners. However, many areas that may need suppression would receive none.

Most opportunities for coordination of suppression between States and within State municipalities and townships would be lost. Depending on the overall organization of suppression efforts, communities and individual landowners may have reduced opportunities for participation in the decisionmaking process. Increased losses of timber and shade trees would be expected to occur.

Isolated infestations that remain untreated would be expected to expand through natural spread of the insect. Depending on the local environmental and physical conditions, the expansion may be rather slow or quite rapid. After untreated populations build to defoliating levels, there will be losses of shrubs, ornamental trees, and timber, and increased insect nuisance.

As impacts on timber and ornamental shade trees increase in the infested areas, and as the isolated infestations increase in number and size, the USDA Forest Service and APHIS will have difficulty in meeting statutory authorities contained in the Cooperative Forestry Assistance Act of 1978 and the Plant Quarantine Act of 1912, as amended.

Chemical Insecticide Treatment

The chemical insecticide treatment alternative would result in funding of proposals using chemical insecticides such as carbaryl, trichlorfon, diflubenzuron, and acephate. These insecticides have successfully achieved the desired objectives in previous suppression and eradication projects, and are registered by the EPA for application against the gypsy moth.

The chemical insecticides will meet project objectives. Because the mode of action of most of these insecticides is by contact, they take effect in a matter of hours after application, and subsequently provide a minimum of 70 percent host foliage protection, and a 90-percent reduction in the number of larvae present and residual egg masses. Further, they can be applied over a wide timeframe during the gypsy moth larval phase and still be effective in reducing gypsy moth populations, though adequate foliage protection may not be achieved.

Implementation of this alternative will provide immediate relief from the presence of gypsy moth larvae in communities and recreation areas. Potential allergic reactions associated with larval droppings and the hairs on gypsy moth larvae will be reduced.

The following matrix compares human health risks with the realistic and worst case doses (developed in Appendix F) that could occur through the aerial application of the four chemical insecticides. It compares the relationship of dose to the ADI (Acceptable Daily Intake), possible teratogenic effects (birth defects), possible systemic effects (such as cholinesterase depression) and cancer. The dose that an individual might receive is evaluated in terms of the levels of safety used by EPA to derive an ADI. The risk of cancer, presented as one chance in a million, has been suggested as a benchmark for this risk analysis. Comparative levels for other risks are presented in Table 17 of Appendix F. It is up to the decisionmaker to determine if this level of cancer risk or the EPA safety factors are acceptable risk levels when weighed against the benefits of suppression or eradication projects.

Is the Realistic Dose Below or Equal to:	Acephate	Carbaryl	Diflubenzuron	Trichlorfon
EPA ADI	Yes	Yes	Yes	Yes
EPA Safety Factor for Terata NOEL	Yes	Yes	Yes	Yes
EPA Safety Factor* for Systemic NOEL	Yes	Yes	Yes	Maybe
Cancer Risk of 1 in a million range	Yes	Yes	Yes	Yes

Is the Worst Case Dose Below or Equal to:	Acephate	Carbaryl	Diflubenzuron	Trichlorfon
EPA ADI	No	No	Yes	No
EPA Safety Factor for Terata NOEL	Yes	Yes	Yes	Yes
EPA Safety Factor* for Systemic Noel	Maybe	Maybe	Yes	Maybe
Cancer Risk of 1 in a million range	Yes	Yes	Yes	Yes

* For three of the insecticides, the expected doses to the general public may exceed the safety factors established by the EPA. These doses occur for exposures which include a dietary component that contains insecticide residue. If an individual consumed produce from a store or ate preserved food and drank water from unexposed water sources (city water, well, or covered spring), then all the doses would be below the NOEL for systemic effects by levels exceeding EPA's safety factor.

The most acute impacts to human health are associated with doses that could be received from exposure to insecticide spills (aircraft and truck). These health effects could range from something as mild as headaches, to serious neurological effects and even death depending upon the length of exposure and how quickly medical attention is provided. Without a doubt, accidental exposures of this type can be extremely hazardous. The risk analysis (Appendix F) identifies these types of effects for all of the insecticides, with the possible exception of diflubenzuron. The probability of accidental spills resulting in realistic doses are estimated to be: 1) 1 truck spill on land for every 93,000 vehicle trips; 2) 1 truck spill in water for every 833,000 vehicle trips; 3) 1 aircraft spill on land for every 1,960 aircraft loads; and 4) 1 aircraft spill in water for every 17,554 aircraft loads. The probabilities of accidental spills resulting in worst case doses are estimated to be 1,000 times lower than those described above.

Additional comparative effects of registered insecticides commonly used in USDA gypsy moth suppression and eradication projects are presented in Table 2. The chemical insecticides as a group are broad-spectrum insecticides that will affect some nontarget insects in the treatment areas, including gypsy moth parasites and pollinators (especially bees). The degree to which these nontarget insects are adversely affected depends on the insecticide and particular formulation used, and mitigating measures implemented.

The Division of Agricultural Sciences, University of California, categorized the relative toxicity of pesticides to honeybees based on laboratory and field tests (Atkins et al. 1981). Using their categories, the chemical insecticides used in gypsy moth suppression and eradication projects ranged from highly toxic to relatively nontoxic.

For example, the insecticide formulations Sevin 80S® (carbaryl) and Orthene® (acephate) are rated as highly toxic to honeybees, with severe losses expected if used when bees are present at treatment time or within a day thereafter. The formulation Sevin 4 Oil® (carbaryl) is rated as being moderately toxic, and can be used around bees if dosage, timing, and method of application are correct, but should not be applied directly on foraging bees or to the hives. The insecticides Dimilin® and Dylox® are rated as relatively nontoxic and can be used around bees with minimal injury.

In December of 1982, the EPA reviewed a number of studies submitted by Union Carbide in support of revised honeybee labeling for Sevin XLR. Based on work by Atkins et al, EPA concluded that Sevin XLR is highly toxic to honeybees exposed to direct application.

Table 2. Comparative effects of registered insecticides used in USDA gypsy moth suppression and eradication projects.*

Characteristics	Pesticides						
	Diflubenzuron	Trichlorfon	Carbaryl	Acephate	Bacillus thuringiensis	Nucleopoly-hedrosis virus	Disparlure
I. Biological Efficiency							
1. Contact poison	0	X	X	X	0	0	0
2. Stomach poison	X	X	X	X	X	X	0
3. Rapid larval knockdown and mortality	0	X	X	X	0	0	0
4. Foliage protectant 1/	X	X	X	X	X	X	0
5. Ovicidal activity	X	0	0	0	0	X	0
6. Population control	X	X	X	X	X	X	X
7. Pre-budbreak control	X	0	0	0	0	0	0
8. Mating disruption	0	0	0	0	0	0	X
II. Economic Feasibility							
1. Tolerance established on agricultural crops	X	X	X	X	0	0	0
2. Dosage lb. a.i./acre	.03-.06	1.0	1.0	0.5	8-16 2/	25-125 3/	1.8 4/
3. Number of applications 5/	1/yr.	1	1	1	1+	2	1
4. Insecticide cost per acre (1983) 6/	Low	Medium	Medium	Medium	Medium	High	Medium
III. Environmental Effects							
1. Residual activity on foliage (10 days)	X	0	X	0	0	0	X
2. Half-life:							
--water (1-2 days)	X	X	X	0	X	X	0
--soil (.5-1 week)	X	X	0	X	X	X	0
3. Adverse effects on nontarget insects:							
--parasites & predators	0	X 7/	X	X	0	0	0
--pollinators	0	0	X	X	0	0	0
4. Adverse effects on wildlife as a group	0	0	0	0	0	0	0
5. Adverse effects on aquatic organisms:							
--invertebrates	X	0	X	X	0	0	0
--fish	0	0	0	0	0	0	0
6. Causes temporary territory abandonment by birds	0	X	X	X	0	0	0

1/ Foliage protection would be achieved by definition when tree refoliation was prevented.
2/ *Bacillus thuringiensis* may be applied at 8-20 Billion International Units per acre per application.
3/ Nucleopolyhedrosis virus is applied at 25-125 million gypsy moth potency units per acre application depending upon natural virus levels in the target area (not currently available for operational use).
4/ Hercon Luretape applied at forty 2-square inch tapes/acre is equivalent to 1.8 grams disparlure/acre.
5/ Eradication treatments involve at least 2 applications.
6/ Low - $9/acre, medium - $9-15/acre, high - $15/acre/application including application and administrative costs.
7/ May temporarily reduce Tachinid fly population.

* NOTE: X = observed effect

0 = no observed effect

Similarly, the persistence of the chemical insecticides in the physical environment also varies according to the individual insecticide and formulation used. However, as described in Table 2, these insecticides as a group are short lived except for diflubenzuron and carbaryl, which have residual activity on foliage of at least 10 days.

Implementation of this alternative will not alleviate the concerns of individuals questioning the use of the chemical insecticides. However, the use of a public involvement and notification program can help minimize these concerns so that individuals residing in treatment areas are aware of the proposed treatment and the available scientific data regarding possible adverse human health effects.

The cost of chemical insecticides for gypsy moth suppression and eradication projects ranges from $3 to $7 per acre per application for material. Total costs, including material, pesticide mixing, loading and application, may range from $10 to $15 per acre per application depending on the chemical used, the number and size of treatment areas, and contract requirements. Cost of ground applications range up to $50 per acre or more depending on these same factors.

Biological Insecticide Treatment

The biological insecticide treatment alternative would result in funding of proposals using biological insecticides. The biological insecticides considered and evaluated during the environmental analysis were formulations of Bacillus thuringiensis (B. t.) and the gypsy moth nucleopolyhedrosis virus (NPV). Both are registered by the EPA for application against the gypsy moth. The gypsy moth NPV, however, is currently undergoing field tests and is not ready for operational use at this time. The comparative effects of insecticides commonly used in USDA suppression and eradication projects are presented in Table 2.

The biological insecticides are considered as environmentally safe. Two reports of B. t. related infections have been recently brought up and are discussed on page 68. These involve an accidental splashing of B. t. into a worker's eye and an accidental puncture wound in the finger with a B. t. contaminated laboratory needle. It is unclear whether there is any implication from these reports for the B. t. variety used in gypsy moth projects. The possibility of accidental exposure to B. t. from aircraft and truck spills is real. Since no true no effect levels (NOELs), or ADIs have been established for B. t., it is probable that there may not be any serious health effects from exposure to accidental spills. The probabilities of aircraft and truck spills occurring are the same as those

discussed for the chemical insecticides. Neither B. t. nor gypsy moth NPV has been shown to adversely affect fish, birds, mammals or most nontarget insects. However, B. t. will affect other lepidopterous larvae if they are present in project areas.

The registered NPV product has a shorter residual life in the environment than naturally occurring NPV in gypsy moth populations. The biological insecticide treatment alternative best minimizes adverse impact on soil, air, water, and humans.

The effectiveness of biological insecticides is dependent on proper application timing. The efficacy of a biological insecticide is also more dependent upon weather conditions, especially rain, than chemical insecticides. Unlike chemical insecticides, biological insecticides must be ingested by gypsy moth larvae to be effective. Since 1st instar larvae feed very little, it is the 2nd and 3rd instars that are most susceptible to the biological insecticides. Recent work at higher potency rates indicates B. t. may also be efficacious against older instars. As a result of this differential response by various aged larvae, there is only about a 2-week optimal treatment "window" during which application of biological insecticides can be expected to achieve maximum effectiveness. Gypsy moth population reduction and foliage protection can be achieved, but to a lesser extent, if the optimal treatment "window" is missed. Since biological insecticides must be ingested, it generally takes 7 to 10 days before larvae die. During this time some insects may continue to feed to some degree and defoliate the host trees. Should application timing be too far past the optimal "window," management objectives may not be achieved. As a result, the biological insecticide treatment alternative may not provide maximum abatement of insect nuisance in some cases; however, it would alleviate some public concerns associated with the use of chemical insecticides.

Recent advances in the use of B. t. have demonstrated that under many treatment conditions, a single application of 12 Billion International Units (BIU) per acre is as efficacious as 2 applications at 8 BIU per acre. This makes B. t. more economical to use than in the past. Current costs for B. t range from $3 to $5 per acre for material. Total costs including material, mixing, loading, and application generally are under $10 per acre, depending on project size. Where more than one application of B. t. is required, total cost rises proportionally. Costs of ground application are comparable to those for the chemical insecticides.

It is estimated by USDA that gypsy moth NPV, though not operationally available at this time, would cost about the same to use as B. t. However, because NPV currently requires two separate applications, 7 to 10 days apart, total project costs are estimated to be twice that for B. t. except where more than 1 application of the latter is required.

The recent demonstrated efficacy of a single application of B. t. in suppression projects and the reduction in product costs, make B. t. economically efficient to use in some areas. This has positive implications for suppression project benefit-cost analyses. However, both the Pennsylvania Bureau of Forestry and the New Jersey Department of Agriculture experienced problems with B. t. in suppressing high gypsy moth populations. In 1984 where B. t. at 12 BIUs was applied to areas in New Jersey having more than 2,000 egg masses per acre, results were generally poor in achieving foliage protection and population reduction targets. Retreatment of many of the B. t. spray blocks will be necessary in 1985 because of the high egg mass numbers remaining in the 1984 treated blocks (NJDA 1984). Pennsylvania experienced similar problems in their 1983 suppression program. B. t. was applied at either 12 or 16 BIUs with the higher rate used in denser gypsy moth populations. Field evaluations showed that larval mortality ranged between 70 and 93 percent. Final defoliation did not exceed 30 percent, and egg mass populations were reduced compared to the previous year. However, many of the spray blocks that had healthy building populations had sufficient egg mass numbers to justify retreatment in the 1984 spray program (PA Bureau of Forestry 1983).

The need for retreatment of B. t. sprayed areas the following year will significantly influence the benefit-cost analysis of such projects.

The biological insecticide alternative would be justified where special environmental concerns have been identified, and where absolute protection of host foliage and a reduction in gypsy moth populations are not required.

Integrated Pest Management (Selected)

The IPM alternative would result in funding of proposals to cooperating State and Federal agencies to support use of an IPM strategy for gypsy moth suppression and eradication projects. The components of this strategy include quarantines, inspections, biological and/or chemical insecticide application, parasite and predator management, application of the gypsy moth pheromone, release of sterile or partially sterile gypsy moth life stages, and forest stand manipulation.

At this time, parasite and predator management, release of sterile or partially sterile gypsy moth life stages, and forest stand manipulation are undergoing field testing and are not available for operational use.

The IPM alternative would affect the environment only to the extent that the various components are used. The impact on soil, air, and water would depend on the amount of acreage treated with chemical or biological insecticides. Impacts on soil, air and water can be further reduced by careful monitoring of gypsy moth larval populations and treating a minimum number of acres with insecticides to achieve specific goals. Areas where parasites or other natural controls are exerting adequate biological pressure on gypsy moth populations probably would not receive insecticide treatment.

An IPM approach which includes the use of both chemical and biological insecticides and other available components can be expected to achieve the objectives of gypsy moth suppression and eradication projects. In terms of foliage protection and population reduction, IPM as defined in the FEIS may perform somewhat less effectively than the chemical insecticide alternative and somewhat better than the biological insecticide alternative.

The cost of implementing the IPM alternative depends upon the extent to which the various operational components are used. The cost of using biological or chemical insecticides was discussed previously. The cost of using disparlure as a mating disruptant strategy is estimated at about $20 per acre for the disparlure-treated tape applied at 20 g active ingredient per acre. Application of the tape in the grid pattern would require at least $1 to $2 per acre, unless the application was handled as a community project. The cost of aerial application of disparlure-impregnated flakes (20 g active ingredient per acre) is estimated at $20 per acre. 2/ Determination of benefit-cost ratios for disparlure application will require additional field evaluation.

Economic information on the use of sterile or partially sterile gypsy moth life stages suggests that at the cost of current avenues being developed it could be competitive with the use of conventional pesticides. However, it is

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2/ Letter from A. R. Quisumbing, Health-Chem Corporation to Noel F. Schneeberger, NA, S&PF, USDA FS, dated November 2, 1983.

important to point out that this approach will be economically feasible only in extremely low-level populations. The cost of implementing other IPM components is not known at this time.

MITIGATING MEASURES

Procedures, guidelines, and other measures can be implemented to mitigate nontarget effects in suppression and eradication projects that include the use of insecticides. During application, insecticide droplets can settle in nontarget areas. Potential impact in those areas as well as exposure to nontarget organisms, including humans, can be minimized by using the proper type of application equipment, proper calibration of this equipment, adherence to strict standards for site and insecticide selection, and by following the operational plan for the project, including the use of buffer strips where necessary.

State and Federal agency participation in USDA suppression and eradication projects provides for early public involvement in the selection of treatment areas and where appropriate, the identification of exclusion areas, and mitigating measures to be used. Through public involvement and notification, individuals known to be allergic to certain insecticides can be notified and appropriate measures can then be taken to avoid exposure to that insecticide. In order that the public is aware when insecticides will be applied, persons living within all treatment areas will be notified by telephone, local newspaper, local radio, individual letter, and/or personal contact as to treatment dates. Users of public recreation areas will be notified in parks or campgrounds. Potential exposure to insecticides is greatest for individuals involved in the actual mixing and application. Proper protective clothing and safety procedures will minimize any risk to individuals involved in these tasks. Safety plans will provide for contingencies, such as pesticide spills and worker exposure.

Specific mitigating measures for suppression and eradication projects will be developed and subsequently implemented on a case by case basis as identified through site-specific environmental analyses, and documented in accordance with NEPA.

A general discussion of treatment area and insecticide selection considerations, application procedures, and monitoring follow. Specific methods and procedures will be identified during site-specific environmental analyses for proposed suppression and eradication projects as necessary.

Treatment Area Selection

In general, public involvement at the community, township, county, and/or State level is an integral part of the treatment-area selection process. Local news media and public meetings are used to inform the public that financial and technical assistance for suppression and eradication projects are available.

Responding to requests from the local level, the State conducts field evaluations to determine if the proposed suppression projects meet the necessary criteria for treatment. Field evaluations of proposed treatment areas include assessment of egg mass size, numbers, and viability; previous defoliation; and land use category. Some States may allow individuals or local residents the option of not participating in proposed suppression. This option is based on State policy or law. Local residents decide whether or not to participate in a cooperative gypsy moth suppression project for those potential treatment areas that meet specific State criteria.

Treatment-area selection in APHIS eradication projects, for Federal and State and private lands, is tied very closely to biological evidence of where gypsy moths are present and reproducing. Highly effective adult male pheromone traps supplemented by larval traps and visual surveys for egg masses provide this biological information. These data, along with the potential for buildup and spread from such areas and environmental impact, are considered before proposing areas for eradication treatment. Individuals do not have a choice of electing to opt out of an eradication project. Cooperating agencies are advised to use the local news media and public meetings to involve the public in the development of these projects.

Minimum treatment area selection criteria are the same for suppression projects on Federal and State and private lands.

Insecticide Selection Application Procedures

Where insecticides are proposed for use, specific selection for that project will be addressed in site-specific environmental analyses documented in accordance with NEPA.

The insecticide selection process considers the project objectives, environmental sensitivities of proposed treatment areas, and the biological and economic efficiency of each insecticide. In addition:

(1) the insecticide must be registered with the EPA for use on the proposed site.

(2) the method of application must conform with label specifications.

The ultimate fate of insecticides released in the atmosphere depends on the insecticide and its formulation, the type of equipment used to apply the material, and the atmospheric conditions during the time of application. The operational plan developed for specific gypsy moth suppression and eradication projects will contain measures designed to maximize insecticide deposition on the target area. Insecticides will be applied in accordance with applicable laws and regulations.

Most gypsy moth suppression and eradication projects will be undertaken using single- or multiple-engine fixed-wing aircraft, or helicopters. Only aircraft that are highly maneuverable and can operate at slow airspeeds close to the tree canopy are used. Precise control of insecticide application is necessary. Pilots can more easily identify hazards and treatment boundaries, and make the necessary insecticide shutoffs. Low-elevation application directly over treatment areas minimizes insecticide drift out of and within the target areas. In addition, observer planes can be used on projects to direct the aerial applicators to the treatment areas, to notify pilots of exclusion areas (no treatment), and to monitor delivery of the insecticide--its release from the aircraft, deposition, and drift. In this way, any mechanical problems can be identified quickly and adjustments can be made in the applications, or the project can be shut down if the insecticide is not falling in the target areas. In areas where Federal Aviation Agency regulations prohibit low-level flying, waivers can be sought to make low-level applications.

Individual treatment areas may range from several acres to several thousand acres depending on the type of project and its location (residential areas vs. forested areas). Treatment areas may include recreational sites, selected high-value forest stands, residential areas including suburban and rural residential areas with sufficient gypsy moth host-type where the insect may create a local impact, including isolated infestations. Consequently, there should be few, if any, treatment exclusion areas within most treatment areas. Treatment and exclusion areas, where appropriate, are identified on large-scale maps that will be used to orient the aerial applicators. If necessary, treatment areas can be designated with helium-filled weather balloons or some other technique that will be highly visible from the air. Treatment exclusion areas will be observed by aerial applicators during pretreatment overflights of all areas.

Insecticides will be applied only when weather conditions favor effective insecticide penetration and dispersal into the target areas. Aerial suppression and eradication projects adhere to the following general guidelines:

(1) To minimize drift, application of insecticide will be made when the wind speed does not exceed 10 mph.

(2) Generally, insecticide application should not be attempted when temperatures exceed 80°F. High temperatures can cause excessive evaporation of the insecticide suspension before it reaches the target. The amount of evaporation depends on the type of insecticide being used. Inversion layers may form in the air when temperatures rise and prevent insecticide deposition.

(3) Insecticide application will be suspended when rain is imminent. After rain, insecticides will be applied only when the target foliage has dried sufficiently.

(4) The treatment will be suspended whenever the insecticide does not appear to be settling in the target area.

Most insecticide treatments are applied in the early morning (4:30 am to 10:00 am) and late afternoon-early evening hours (4:00 pm to 9:00 pm), as this is when atmospheric conditions generally are the most favorable for maximizing insecticide deposition in treatment areas. Occasionally insecticide applications may continue throughout the day, so long as conditions are favorable.

Where aerial application of insecticides is not appropriate, ground equipment may be used. Specific ground application guidelines will be developed on a site by site basis in order to mitigate unacceptable environmental impacts. These specific guidelines will be identified during site-specific environmental analyses for suppression and eradication projects and conducted in accordance with NEPA.

Beekeepers in and adjacent to treatment blocks will be notified as to the time of treatment, insecticide to be used, and the availability of pollen traps if applicable. All residents or persons visiting a treatment area are notified in advance of treatment so that they may leave or stay indoors at the time of treatment depending on their personal health and desires.

Monitoring Procedures

Monitoring is a continual process taking place before, during, and after treatment application. Specific monitoring techniques used to determine gypsy moth population levels and subsequent candidate treatment areas, to evaluate proper insecticide deposition and to evaluate project efficacy are identified in the cooperating State

or Federal agency proposals. In suppression projects, State and Federal cooperators usually begin the process by conducting egg mass surveys to determine treatment areas. The egg masses in selected areas may be monitored for winter survival and the effects of parasites and predators to determine if treatment still is needed. In planning eradication projects, sampling of other gypsy moth life stages is necessary to determine whether a potential low level, reproducing population exists.

During insecticide application, spray deposit cards or a similar technique can be used to check deposition and drift. Observer aircraft may be used during aerial insecticide applications to monitor spray dispersal. Daily weather measurements of temperature, wind speed, and relative humidity generally are made on site, and subsequent communication with weather stations to help ensure that insecticide application is made under the proper weather conditions.

Following suppression activities, project personnel generally visit a representative sample of treatment blocks to assess larval mortality. Later in the summer, aerial defoliation estimates will be conducted statewide. Ground estimates may be made if necessary. In the fall, egg mass counts are conducted in selected areas to measure population reduction. Because of extremely low populations found in eradication projects, treatment efficacy is monitored by larval traps and/or male moth pheromone traps.

AFFECTED ENVIRONMENT

HOST VEGETATION

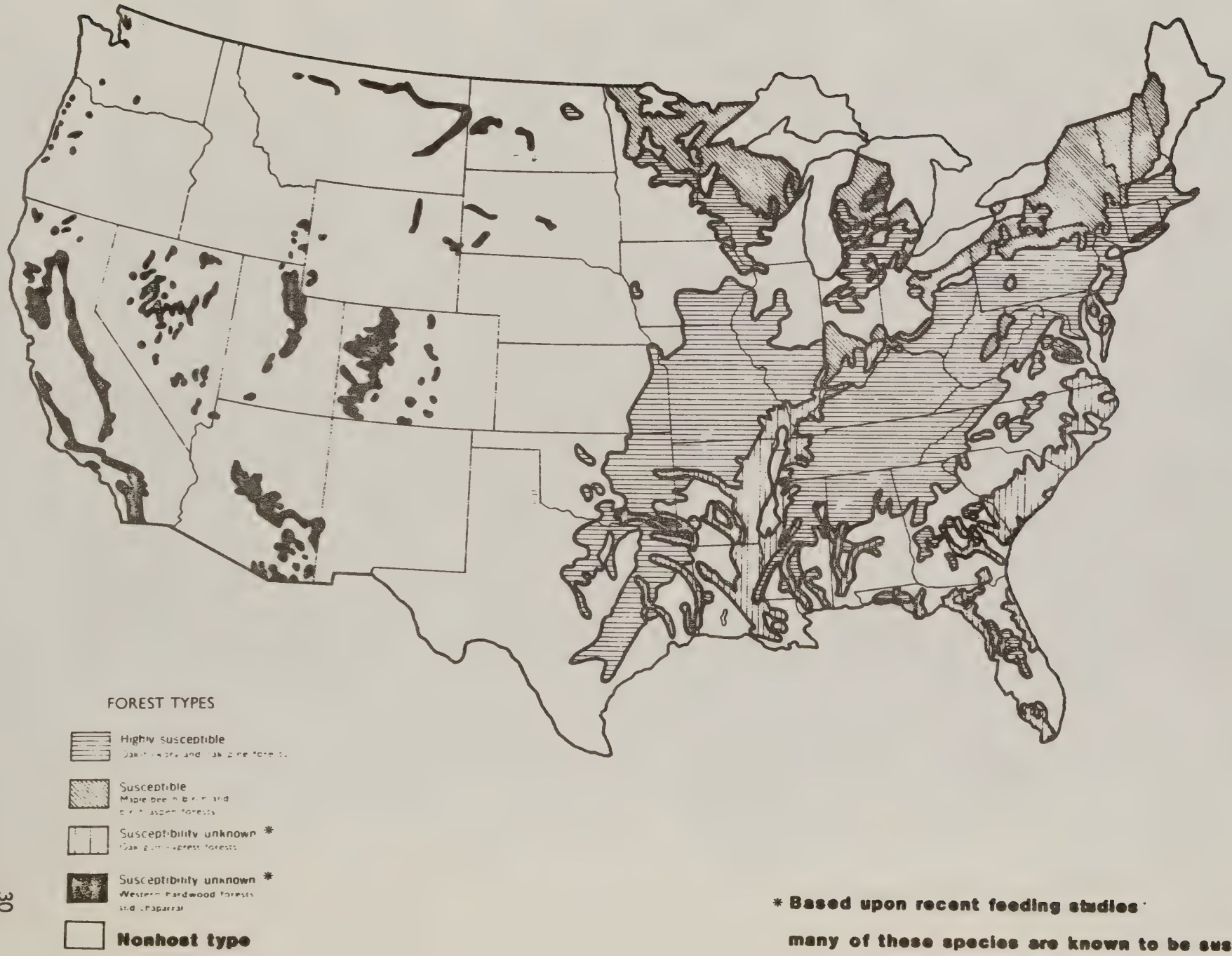
The gypsy moth feeds on more than 300 species of trees and shrubs (USDA 1981a). Figure 1 depicts the location of forest host types in the continental United States that are susceptible to gypsy moth infestation. Preferred hosts are oak species, especially white oak. Additional hosts include apple, basswood, gray and river birch, sweetgum, hawthorn, poplar, beech, willow, and other oaks. Less desired but still attacked are black birch, yellow birch, paper birch, cherry, hemlock, cottonwood, elm, sassafras, spruce, and pine. Older gypsy moth larvae feed on the foliage of several species that younger larvae normally avoid, particularly hemlock, pine, and spruce. The gypsy moth avoids ash, balsam fir, butternut, black walnut, catalpa, red cedar, flowering dogwood, American holly, locust, sycamore, tulip (yellow) poplar, and shrubs such as native laurel, rhododendron, and arborvitae. However, in outbreak situations, gypsy moth larvae will feed on almost all vegetation.

Recent gypsy moth feeding studies with late 3rd-instar larvae were conducted on plant species common to California (Edwards and Fusco 1981). Study results indicate that the following host types appear to be susceptible to gypsy moth: grand fir; acacia; red alder; apricots; manzanita; beefwood; California hazel; escallonia; eucalyptus; hakea; sweetgum; ironwood; photinia; white spruce; Norway spruce; Coulter pine; Jeffrey pine; Monterey pine; Digger pine; Pinus halpensis; Pinus thunbergiana; Douglas-fir; pyracantha; blue oak; California black oak; California white oak; cork oak; hawthorn; lemonade berry; weeping willow; redwood; western redcedar; and western hemlock. As gypsy moth populations continue to spread to the South and West, the number of plant species known to be susceptible will undoubtedly increase.

POTENTIAL TREATMENT SITES

Permanent residences often occur throughout the range of susceptible host vegetation. On private land, permanent and seasonal houses are located in suburban/urban areas, rural residential areas, and in forested areas. Entire developments and forested communities are designed and constructed to maintain a forest setting. On public lands, forested areas are developed into recreational areas, campgrounds, picnic areas, hiking trails, and scenic areas. Visitors include campers, canoeists, anglers, hikers, and others. Most of the recreational use is concentrated around water, scenic areas, or parks.

Figure 1. Forest types susceptible to gypsy moth.



Gypsy moth suppression projects may be conducted in urban, suburban, or rural communities as well as in unpopulated forests. In suppression projects, some States may allow individuals or residents the option of not participating in proposed suppression. This option is based on State policy or law.

APHIS eradication projects may require treatment of infestations in areas that range from highly populated urbanized areas to rural or uninhabited sites. In addition, infested sites may range from generally open areas occupied with shrubs and those with occasional ornamental trees to highly forested communities or uninhabited forests. In all of these sites, the public involvement process will allow individuals an opportunity to provide input into the decisionmaking process. However, since the goal of the project is eradication, individuals do not have the option of having their property excluded from treatment.

NONTARGET ORGANISMS

There are many species of fish, mammals, reptiles, birds, and amphibians that inhabit the various forest types susceptible to gypsy moth infestations. Other nontarget organisms occurring in gypsy moth-susceptible host types include terrestrial and aquatic insects; pollinators, including bees needed for honey production; gypsy moth parasites and predators; and soil organisms. Some insect populations may be reduced temporarily by insecticides used for gypsy moth suppression.

Gypsy moth suppression and eradication projects are not expected to adversely affect threatened and endangered species that may be found within treatment areas. However, pursuant to the Endangered Species Act of 1973, consultation procedures will be initiated with the USDI Fish and Wildlife Service, when appropriate to identify any projects that may affect threatened and endangered species. Since suppression and eradication activities may take place anywhere in the United States where there are susceptible hosts, evaluations of threatened and endangered species will be addressed in site-specific environmental analyses, and conducted in accordance with NEPA.

GEOGRAPHY

Gypsy moth will continue its natural spread south and westward into States not now generally infested.

The insect is now permanently established in all or parts of the following States: Connecticut, Delaware, Maine, Maryland, Massachusetts, Michigan. New Hampshire, New

Jersey, New York, Rhode Island, Pennsylvania, Vermont, Virginia, and West Virginia.

Because the geography and other related physical factors of these areas vary considerably, they will be addressed during site-specific environmental analyses, and conducted in accordance with NEPA for the particular areas proposed for treatment.

APHIS eradication projects will be conducted in areas where the insect has been introduced artificially. These artificial introductions may occur in areas throughout the continental United States and Hawaii. Treatment areas may occur in any geographic or physical setting, from low coastal to mountainous areas. Because these factors may vary considerably, they will be identified during site-specific environmental analyses conducted in accordance with NEPA for the particular areas being considered for eradication treatment.

ENVIRONMENTAL CONSEQUENCES

NO ACTION

This alternative would not necessarily eliminate gypsy moth suppression or eradication activities. State agencies, local communities, and individuals may undertake projects without Federal financial assistance. Under this alternative, the following general effects can be expected.

In the infested areas of the Northeast, if no action is taken by either State or local agencies or by individuals, gypsy moth-caused tree defoliation and subsequent mortality will occur.

Gypsy moth populations will increase and then collapse--a cycle of several years. Impacts of unimpeded gypsy moth-caused tree defoliation include tree mortality, reduced growth, and changes in forest stand composition. These effects will be dramatic in the short term and their effects will last for many years. Several case studies illustrate the point. A study by Kegg (1972a) found that 2 years of heavy defoliation in northern New Jersey in 1969 and 1970 killed 63 percent of the oaks in the study area. Stephens and Hill (1971), studying changes in Connecticut forests from 1959 to 1970, found that repeated defoliation increased oak mortality by 50 percent.

A reduction in radial growth also is associated with defoliation by forest insects (Kulman 1971). In Europe, reductions of 15 to 67 percent were observed following severe gypsy moth outbreaks (Mirkovic and Miscevic 1960; Kondakov 1963). Similar observations have been made in the United States (House 1960; Minott and Guild 1925).

It is generally believed that gypsy moth activity tends to influence forest stand composition (Campbell and Valentine 1972; Stephens 1971). This might be perceived as being beneficial in some areas by reducing favored food hosts. However, Bess et al. (1947) stated that "frequent severe defoliation will generally create conditions highly favorable to future epidemics."

Several years of heavy gypsy moth defoliation may have an adverse impact on existing wildlife (Campbell 1979). It is logical to assume that animals that normally inhabit forests will be adversely affected by several years of gypsy moth-caused defoliation and subsequent tree mortality.

In the infested areas of the Northeast, where there is a complex of parasites and predators, it is possible that these organisms, as well as other natural controls such as wilt disease, will eventually reduce gypsy moth populations. However, this will not occur immediately, and dramatic effects such as those discussed earlier will occur.

Streams shaded by forest trees are inhabited by aquatic plants and animals. Defoliation removes the shade from those streams, producing light and heat greater than the average to which the aquatic life is adapted. In the year of defoliation, these conditions last until the trees refoliate. Under several years of successive defoliation, such conditions, while only temporary the first year, may become more permanent characteristics if tree mortality changes the composition of tree species.

Heavy gypsy moth populations may affect water quality and quantity. Frass excreted by larvae is considered both a nutrient dump and water pollutant. Observations on the impact of frass are scarce. Turner (1963) stated: "Heavy defoliation produces hundreds of pounds of frass which soon is washed into the water by rain. The effect on the quality of water is immediate in small reservoirs. Moreover, the nutrient elements in the frass increase the growth of algae in water, creating an additional problem of longer duration." A similar observation was reported for New Jersey reservoirs (Kegg 1972b).

Where gypsy moth-caused defoliation and subsequent tree mortality is severe, there is less vegetation to intercept rainfall and to impede the movement of ground water. This could increase streamflow but at an added cost. Water quality might be lowered due to increased runoff of soil and soil nutrients previously held in place by vegetation now destroyed by gypsy moth. Defoliation along streams may result in increased water temperatures that not only affect existing aquatic organisms but also influence plant compositions along stream banks. While the impact of gypsy moth on soil and plants other than trees has not been

studied in detail, it is known that when the forest floor is opened to unaccustomed heat and light, changes in forest soil and plant life occur (Bess et al. 1947; Kegg 1972b).

Gypsy moths invading homes can seriously affect individuals with a fear of insects. Some individuals actually have cut down all trees on their property to avoid the nuisance of the insect. Others have avoided the use of summer homes during the period in which larvae are feeding.

Recent medical studies have confirmed that the hairs on gypsy moth larvae can cause skin rashes and welts (Shama et al. 1982; Beaucher and Farnham 1982; Tuthill et al. 1984). The intensity of allergic reactions in some individuals surprised researchers. Some individuals could not sleep at night and required injection of corticasteroids for relief after simple antihistamine and topical treatments failed (Beaucher and Farnham 1982).

Conditions experienced in Pennsylvania during the 1973 outbreak are reported in this summary (PA DER 1973):

> Carbon and Schuylkill Counties were considered by local officials to be disaster areas. The mayor of Tamaqua declared a state of emergency as caterpillars invaded the town and the water supply reservoir. Farmers in outlying areas reported corn and alfalfa being eaten. A new form of "control" was witnessed near Tamaqua as a homeowner started a fire under trees in his yard. The flame and heat did a good job on the trees as well as the gypsy moth. Signs of all descriptions appeared along Schuylkill County roadsides denouncing government officials from the local level to Washington. In some areas, housewives reported that caterpillars had invaded homes and began eating house plants. Golfers reported that putting greens were literally moving with migrating caterpillars. People on vacations checked into untreated resorts but left the next day.

Even the natural disease agents affecting gypsy moth may be unpleasant. The disease-killed larvae hang from their sheltered places on houses; their bodies rupture and the rotted fluid contents spill out, staining homes. Bacteria grow in the fluid, making the stench of a diseased population detectable from a distance.

It is difficult enough for people to live with the larvae and frass of a gypsy moth outbreak. Yet they also must endure the experience of having the trees around them stripped of their leaves in June and July. Hardwood trees losing about two-thirds of their foliage generally develop another set of leaves (refoliate), beginning about mid-July. Such leaves usually are smaller, off-color, and fewer in

number than the original leaves. In addition, refoliation decreases valuable sugar reserves needed to sustain the tree through the following spring and winter.

Two important impacts resulting from defoliation are reduced benefits and enjoyment from the trees. These two impacts blend but it is convenient for this discussion to treat them as separate entities.

The direct benefits derived from trees that are in full foliage include cooling and humidifying effects, shade, reduced sound levels, privacy, and shelter from wind. These benefits often are taken for granted, and it is when the leaves are gone that many people realize clearly the benefits that trees provide.

Gypsy moth-caused defoliation drastically alters the forest homeowner's environment. One purpose for living among trees is to enjoy them; robbed of that enjoyment by the gypsy moth, a person may experience decreased values associated with aesthetic surroundings.

In areas of heavy gypsy moth outbreaks, recreational use of the forest and one's own backyard can be curtailed severely due to the presence of larvae and their droppings. Painting of homes must be curtailed in outbreak areas as larvae stick to painted surfaces and congregate in sheltered places--under window ledges, porch roofs, door sills, and eaves. This necessitates repainting damaged areas.

Outdoors, sidewalks are avenues for migrating larvae. Individuals trying to use those surfaces step on larvae. Sidewalks become stained and caked with the remains of trampled caterpillars. Crushing them makes sidewalks both slippery and unsightly.

Larvae also migrate over driveways and roads. Vehicular traffic crushing larvae in an outbreak area can make roads slippery and dangerous, sometimes requiring highway department trucks to sand unsafe roads during July in the Northeast.

If no eradication action is taken against isolated infestations by State or local communities, gypsy moth populations, depending on local site conditions, are expected to thrive and become well established. The longer eradication action is delayed, the greater the opportunity for insect spread from the immediate site of the infestation into the surrounding area.

Regulatory action by APHIS and State agencies still would be implemented in the form of quarantines, inspections and treatment of materials and goods passing out of the quarantined area to stem the artificial spread of the

insect. However, as the infested area enlarges through natural spread of the insect, increased manpower would be needed each year to maintain the quarantine. Additional impacts are presented by Galt (1983), who discusses the potential economic effects of a no action alternative in California.

Under such conditions, gypsy moth populations in areas having susceptible host material may remain at low levels for several years. Eventually, the populations will build to outbreak levels, causing defoliation of susceptible hosts and conditions similar to those described in the infested areas of the Northeast. During periods when population levels are high, expansion of isolated infestation will occur as a result of natural and artificial spread of the insect. This will occur despite the regulatory actions implemented to contain the spread.

So long as no eradication or suppression action is taken by State or local agencies, anxieties concerning the use of insecticides and human health concerns would not occur. However, based on past experience, the presence of large numbers of gypsy moth larvae and associated defoliation on trees and shrubs would trigger public sentiment for nuisance abatement.

If State agencies or individuals attempt suppression, the biological effects of such would depend on the strategy used and would not differ from the effects previously discussed for each treatment alternative. However, suppression activities undertaken by individuals or communities without the benefit of coordination with a Federal/State-administered program may result in the application of more insecticide than is necessary, since most treatment financed by local communities or individuals entails the use of ground application equipment with varying rates of application.

Suppression or eradication activities undertaken by groups of residents seldom provide for involvement by, and notification of, adjacent residents or other people who might be interested in participating. Benefits derived from organizing a coordinated suppression approach, including maximized treatment efficiency and public safety, would not be achieved.

If the no action alternative results in no attempts by State, local communities, or individuals to suppress populations in infested areas, gypsy moth populations will continue to thrive and spread by natural and artificial means throughout all susceptible host types in the United States. The rate of spread can be expected to accelerate as more and more acres of host type become naturally and artificially infested.

Impacts on timber and ornamental shade trees will increase. The USDA Forest Service and APHIS will have difficulty in meeting the objectives of their statutory authorities contained in the Cooperative Forestry Assistance Act of 1978 and the Plant Quarantine Act of 1912 as amended, with regard to the gypsy moth.

CHEMICAL INSECTICIDES

The 1983 ruling by the United States District Court for the District of Oregon enjoined the USDA APHIS program from using the aerial application of carbaryl to eradicate gypsy moth from populated areas of Oregon (Oregon Environmental Council versus Kunzman et al.) until a new EIS was prepared that considers, among other aspects, the health risks of spraying residential areas including the danger to children and others sensitive to chemicals. In addition, issues concerning the health risks of using chemical insecticides in general were identified during the scoping process conducted prior to development of the draft EIS. Many of the same issues were also reiterated in review comment letters to the draft EIS.

As would be expected, USDA found a paucity of documented scientific literature dedicated specifically to the study of the direct or indirect effects to humans from exposure to chemical insecticides used in gypsy moth or similar projects. Studies specific to sensitive populations (children, pregnant women, people sensitive to chemical insecticides, the elderly, and similar cohorts) were lacking. However, several studies involving exposure effects to volunteers and occupationally exposed individuals were available and subsequently used as a basis upon which to evaluate the significance of exposure to people in or near treatment areas.

While the acute effects of the chemical insecticides are well documented and accepted by the scientific community, the chronic or long-term effects of very low exposure levels are a source of uncertainty. The available literature suggests that there is considerable disagreement among scientists regarding the extrapolation of some test results to humans for effects such as mutagenicity, carcinogenicity, teratogenicity, and others. Much of this controversy lies in disagreements of the significance of intra- and inter-species differences in the laboratory subject animals, interpretation of results, and study design.

In order to address issues and concerns regarding use of chemical insecticides, the USDA developed an analysis of human health risks (to both general and sensitive populations) involving the use of acephate (Orthene), carbaryl (Sevin), diflubenzuron (Dimilin), and trichlorfon

(Dylox) in gypsy moth suppression and eradication projects. The analysis relies on existing data where available and uses worst case assumptions where appropriate. This analysis complies with CEQ regulations (40 CFR 1502.22) regarding gaps in relevant information or scientific uncertainty, and is appended to this FEIS (Appendix F). Results of the analysis are summarized and presented in the discussions of the chemical insecticides which follow.

Efficacy and possible nontarget effects and risk to humans of any new insecticides that may become registered for gypsy moth control in the future will be addressed in environmental analyses conducted in accordance with NEPA for all projects for which their use may be proposed.

Worst Case Analyses

In the process of analyzing the environmental and human health risks associated with the use of acephate, carbaryl, diflubenzuron, or trichlorfon, data on such items as environmental fate, human exposure, human epidemiology studies, acute and systemic toxicity, teratogenicity, mutagenicity, and carcinogenicity were evaluated as to their availablity. Council on Environmental Quality regulations (40 C.F.R. 1502.22) require that when an agency is evaluating significant adverse effects on the human environment in an environmental impact statement, they shall always identify any gaps in relevant information or scientific uncertainty. If the information relevant to adverse impacts is essential to a reasoned choice among alternatives, and is not known, and the overall costs of obtaining it are not exorbitant, the agency shall gather the information and include it in the environmental impact statement. However, if the overall costs of obtaining the information are exorbitant or the means to obtain it are not known (e.g., the means for obtaining it are beyond the state of the art) the agency shall weigh the need for the action against the risk and severity of possible adverse impacts were the action to proceed in the face of uncertainty. If the agency proceeds, it shall include a worst case analysis and an indication of the probability or improbability of its occurrence.

The availability of relevant information is indicated below:

Availability of Information	Acephate	Carbaryl	Diflu-benzuron	Trichlor-fon
Acute toxicity	+	+	+	+
Systemic toxicity	+	+	+	+
Epidemiology	-	*	-	-
Teratogenicity	+	+	+	+
Mutagenicity	+	+	+	+
Carcinogenicity	+	+	+	+
Human Exposure	-	+	-	-
Nontargets	+	+	+	+
Environmental Fate				
Animals	+-	+-	+-	+-
Vegetables	+-	+-	+-	+-
Water	+	+	+	+

* One study dealing with birth defects.
\+ Indicates data available.
\- Means no data found
+- Means data found for agriculture situations, but site-specific data for gypsy moth control was lacking.

Obviously, there were gaps in the data that were relevant for evaluating adverse effects to the human environment associated with gypsy moth suppression and eradication projects. Since the epidemiology or human toxicology data was generally lacking, adverse effects were evaluated using risk analysis techniques which are based on using test animal data. This analysis is documented in Appendix F.

In developing the risk analysis, the lack of human exposure data, including information on dermal absorption, and site-specific insecticide residue data for edible meats and vegetables were the major information gaps. In addition, studies were lacking on cumulative and synergistic effects for the four chemical insecticides. Also, there is uncertainty about the risk of heritable human mutations, the assessment of which, is beyond the state of the art. Lastly, uncertainties were identified in existing data about whether trichlorfon, acephate, and 4-chloroainline (a metabolite of diflubenzuron) were carcinogenic, whether carbaryl could form N-nitrosocarbaryl in humans, and how to extrapolate animal data to humans.

The costs of filling these data gaps for a single compound and estimated times to conduct the tests are listed below (costs and time based on personal communication with Jack Warren, DOW Chemical Co.):

	Cost/Test (thousands $)	Time/Test
Epidemiology tests	$300-$500	
Exposure (human)	$100-$200	2 years
- Pharmacokinetics (rat)	$140	1 year
- Pharmacokinetics (mouse)	$140	1 year
Residue Analysis		
Animals	$60-100	1-2 years
Vegetation	$60-100	1-2 years
Oncogenicity (rat)	$440	3 years
Oncogenicity (mouse)	$500	3 years

Obviously, cost of studies to determine cumulative or synergistic effects would be multiples of these costs. For synergistic effects these costs would escalate rapidly as 2, 3, 4, etc. additional chemicals, are added to the study. The pharmacokinetics information is needed if the exposure studies are to be based on urinalysis data. If patch studies are used to determine dermal exposure, then dermal penetration studies in humans would be necessary to determine the percentage of insecticide absorbed into the body. An infinite time period was designated for the epidemiology studies because of the long-time period over which epidemiologists look for effects in cohort studies, or the seemingly infinite number of case-control studies that could be run for specific health responses (including all types of cancer, birth defects, and abortions). Because of the costs and time involved, a worst case analysis was done to assist the decisionmakers in weighing the risk of proceeding with suppression or eradication projects in the face of lacking data.

In the risk analysis (Appendix F), the data gaps for human exposure and residue chemistry were filled by extrapolating from other data sets. A number of assumptions were used to make the extrapolations. A range of assumptions was used to cover both the realistic and "worst case". The specific assumptions are all described in Appendix F. It is important to realize that there is a certain amount of uncertainty in every number used in the risk analysis. Because of this uncertainty, assumptions or procedures were used that would intentionally overestimate risk.

The worst case is defined by the assumptions that are used to determine the worst case human dose values that could occur through normal operations or accidents such as spills. This is only one method for defining a worst case. For example, Crouch et al. (1983) defines upper limits on the cancer risk as the 98th percentile of the distribution of risk. In this case the variance (σ^2) of risk is the sum of the variances for human exposure, for the animal to human extrapolation and for the cancer potency. Since the data needed to calculate these type of probability distributions were not generally available, uncertainties in the data were accounted for in the risk analysis by attempting to overestimate human dose.

Forest Service and APHIS have chosen to define a "reasonable" worst case by trying to estimate dose values that fit the real world, but that would always overestimate risk. For example, the worst case dose value for residents was based on the highest exposure value reported in carbaryl exposure studies. This value was then multiplied times two to account for possible mixing and application errors. Since only a small fraction of the residents actually reported detectable residues, the average dose (50 ppb) would have been 5 times lower than the maximum or worst case value used in this analysis (250 ppb). For residents, who also consume food and water containing insecticide residues, a dietary component of 0.058 to 0.103 mg/kg/day (depending upon the specific insecticide) was added to the worst case dose resulting from direct application. The cummulative worst case dose was then at least 20 times higher than the highest residential dose reported in exposure studies. The worst case dietary doses were orders of magnitude (1000x) higher than insecticide residues reported in marketbasket surveys by FDA. This worst case dose was then compounded with the assumption that each person would receive the maximum dose, when the actual data show that only about 20 to 30 percent of the residents in spray areas actually contact detectable exposures.

Where uncertainty in scientific data existed regarding carcinogenicity of the insecticides (e.g. acephate, 4-chloroaniline, and trichlorfon) the risks were analyzed based on the worst case assumption that the insecticides in question were carcinogenic, or in the case of carbaryl, that N-nitrosocarbaryl would be formed in humans. Furthermore, the cancer model used to measure cancer risk represents a simplified linear model, that by design overestimates risk. The threshold responses, such as NOELs for cholinesterase inhibition and birth defects, also have additional worst case safety margins beyond the dose overestimate. In most cases, the toxicity data that was reviewed reported only the highest dose tested at which no adverse effect was observed.

These highest doses tested were selected as NOELs when actually the true threshold doses or lowest observable adverse effect levels (LOAELs) would be at some higher dose level. The use of the highest dose tested as NOEL was intended to overestimate risk for the worst case analysis.

For the worst case analysis, insecticide exposures that could result from accidents were based on situations that could realistically occur. These included dumped aircraft loads and truck spills. Historical data shows that these type of accidents have finite probabilities of occurrence. There are certainly other worst case situations that would result in more severe exposures or health hazards. These could range from an aircraft crashing into a school bus loaded with children to a meteorite hitting a pesticide storage area. However, such situations were thought to trivialize the worst case analysis because they were beyond the realm of reasonableness and would therefore not provide useful risk information to the decisionmaker.

As has been discussed, the compounding of assumptions results in doses that exceed those that have actually been measured in the field. The cancer risks generated using these doses also represent overestimates that are inherent in the cancer model used. It should be mentioned that EPA and other agencies who implement regulatory policy and set standards may use different cancer models and exposure scenarios than those used in Appendix F. Therefore, the margins of safety identified, and the comparisons to NOELs used for evaluating threshold effects, and the cancer risks calculated for acephate, carbaryl, diflubenzuron, and trichlorfon are relevent to the assumptions used and procedures followed in the risk analysis. Care should be taken if the risks identified in Appendix F are used outside the assumptions and uncertainties described in the risk analysis.

Acephate

General Information. Acephate, trade name Orthene, is an organophosphate compound used as a contact and systemic insecticide. It has a cholinesterase inhibition mode of action (Chevron 1976). It is registered for use to control a broad spectrum of insects on ornamentals, trees, shrubs, and flowers.

Acephate is a white crystalline solid with a very low vapor pressure (2×10^{-6} mm Hg at 25°C) and a very high solubility in water (65 percent).

Fate in Environment. Laboratory studies indicate that acephate is rapidly degraded in soil, primarily due to the action of microorganisms. Field and laboratory studies have shown that acephate rapidly degrades in plants (Chevron

1973). Generally, a 5- to 10-day half-life has been noted in plants (Chevron 1973). Willcox (1973) reported that after applications of up to 0.5 lb per acre, residues in leaves and litter dropped below 0.02-ppm (the limit of detection) in 33 days.

Acephate breaks down relatively slowly in water as the rate of hydrolysis is affected by temperature and alkalinity. The half-life in water at pH 7 and 70°F is about 50 days under laboratory conditions. In natural bodies of water, degradation would be accelerated by breakdown in aquatic vegetation and microorganisms in sediment (Chevron 1975).

From kinetic reaction studies, it has been determined that about 5 to 10 percent of acephate degrades into methamidophos which itself is a registered insecticide for use on certain lepidopterous larvae. Methamidophos is rapidly degraded in soil (Leary and Tutass 1968) and poses no threat for bioconcentration (Chevron 1973). The remaining acephate degrades directly into innocuous salts (Tucker 1972). No other metabolites of toxicological significance have been observed (Tutass 1968).

Toxicology. Acephate will adversely affect some nontarget insects within treatment areas. The effects on nontarget insects from an aerial application of Orthene at 0.5 lb active ingredient per acre for control of the gypsy moth were monitored up to 1 month after treatment (LOTEL 1975). It was concluded that lepidopterous larvae, dipterous larvae, and Hymenoptera--predominantly the family Formicidae--were adversely affected. The order Coleoptera was least affected while dipterous larvae showed the greatest decline in numbers. A knockdown effect observed immediately after treatment affected all orders of arthropods collected; however, populations that were depressed recovered to pretreatment levels within 1 month, and none was eliminated. Acephate has been rated as being highly toxic to honey bees (Atkins et al. 1981).

In laboratory animals, the acute oral LD_{50} of acephate ranges from 866 to 945 mg per kilogram of body weight (mg/kg) (Meister 1983). No observable effect levels (NOELs) have been established in laboratory animals for a variety of effects (see Appendix F, Table 3). Long-term feeding studies in rats (90 days to 28 months) and dogs (2 years) for detection of cholinesterase inhibition activity in plasma, red blood cells, and the brain have established a NOEL of 0.25 and 0.75 mg/kg/day, respectively. A NOEL of 2.5 mg/kg/day was established in dogs (2-year feeding study) for more pronounced systemic cholinesterase effects. NOELs for teratogenic effects (birth defects) have been estimated to be above 10 and 200 mg/kg/day in rabbits and rats, respectively.

The acephate exposure levels to animals that are associated with gypsy moth suppression and eradication projects have been estimated in the risk analysis (Appendix F, pp. F-32 to F-36). The risk analysis uses the goat and the rabbit as representatives of large and small animals (wildlife, livestock, domestic) since both have a high surface area to body weight ratio. Based upon the dermal exposure and oral dose levels identified in Appendix F (p. F-36) a total dose (realistic and worst case) can be estimated. For large animals (represented by the goat) the realistic dose is estimated to be 0.23 mg/kg/day [(0.02 mg/kg/day + 0.29 mg/kg/day) x 0.75]. The worst case dose is estimated to be 2.31 mg/kg/day [(0.2 mg/kg/day + 2.88 mg/kg/day) x 0.75]. For small animals (represented by the rabbit) the realistic dose is estimated to be 0.26 mg/kg/day [(0.04 mg/kg/day + 0.30 mg/kg/day) x 0.75]. The worst case dose is estimated to be 2.55 mg/kg/day [(0.4 mg/kg/day + 3.0 mg/kg/day) x 0.75]. The estimated realistic doses for both large and small animals are about equal to the lowest NOEL value established for the rat and the dog (Appendix F, Table 1). This NOEL value is for cholinesterase inhibition, a readily measurable, low dose response that is characteristic of organophosphate insecticides.

However, the realistic doses are about 40 times lower than the highest dose tested for establishing a teratogenicity NOEL in rabbits (Appendix F, Table 1). The worst case doses in large and small animals range from 3 to 10 times above the NOEL value for cholinesterase inhibition, and are equivalent to the NOEL value in dogs for systemic effects. The worst case doses are 4 to 80 times below the teratogenicity NOEL for rabbits and rats (Appendix F, Table 1). It is possible that the realistic doses could cause a measurable and temporary depression of cholinesterase activity in some animals if exposed to those doses over a long period of time. Prolonged exposure to worst case doses could possibly cause measurable cholinesterase inhibition and some systemic effects. It is estimated that the probability of a worst case dose being applied is 1.8×10^{-3} or 1 worst case dose for every 556 realistic doses. The realistic and worst case doses for animals do not make adjustments for the shielding effect of fur, nor do they take into account the ability of the animal to metabolize, detoxify and excrete acephate. This suggests that even realistic doses are overestimates.

Decreased cholinesterase activity in some animals has been shown. In 1976, acephate was applied at 0.5, 1.0, or 2.0 lb a.i./acre on Wallowa-Whitman National Forest in Oregon. The effect of acephate spraying on brain cholinesterase activity was evaluated in 14 passerine species. All dosages caused marked, widespread, and prolonged brain cholinesterase depression in passerine birds (Zinkl et al. 1979). Also,

postspray bird census data suggested that 2 species of birds may have left the area following the treatments (Richmond et al. 1979). In 1977, acephate was applied at 0.5 lb a.i./acre on forested lands in Idaho for western spruce budworm control. Eleven avian species evaluated showed brain cholinesterase depression (Zinkl et al. 1980).

No human health problems have been demonstrated in the various field development programs in which acephate has been used. When used in accordance with label instructions, acephate poses no health hazard to persons formulating, spraying, or working in treated areas (Willcox and Coffey 1977). For example, a monitoring and medical study was conducted after several men were occupationally exposed to acephate in a plant where the material was being produced, and in a lab where large batches were formulated (Pack 1972). Urine samples were monitored for acephate and metabolites. Concentrations up to 5 ppm were detected in the urine and no adverse health effects were observed. Effects on blood cholinesterase levels, a sensitive indicator of organophosphate exposure, were not detected.

For members of the general public exposed to acephate in gypsy moth projects, the risk analysis (Appendix F) indicates that all realistic doses (exclusive of accidents), and many of the worst case doses are all below the ADI (acceptable daily intake) established by EPA. For occupationally exposed individuals the mixer/loaders might receive a realistic dose that slightly exceeds the ADI, although the dose is close enough to the ADI to be a result of rounding error in the calculations. The only exposure scenarios that exceed the ADI are associated with worst case doses to the occupational group, and to members of the general public who consume a daily diet of meat, fruits, vegetables and liquids containing acephate residues. The question is whether or not these doses are outside an acceptable margin of safety.

Appendix F (p. F-87 to F-90) discusses the relationship of these doses to the lowest NOEL values in Table 1 (p. F-114), and to the data points and assumptions made to carry out the risk analysis. Briefly, the lowest NOEL values used in the comparison were based upon measurable depression in cholinesterase levels in red blood cells, plasma, and the brain. The NOEL does not represent the onset of clinical symptoms characteristic of organophosphate intoxication. Cholinesterase inhibition is the first measurable response to acephate exposure and one that can be treated when clinical symptoms appear. However, as is typical of cholinesterase depression, clinical symptoms generally do not manifest themselves until depression exceeds 50 percent.

Cholinesterase inhibition NOEL values (Appendix F, Table 1) range from 0.25 mg/kg/day in the rat to 0.75 mg/kg/day in the dog. Applying the higher NOEL to the doses in question results in margins of safety that approach the 10 safety factor used by EPA to derive the ADI. As discussed in Appendix F (p. F-90) the dose attributable to the dietary component is what drives the worst case dose above the ADI. Pesticide residue data gathered for all food types by the FDA (Food and Drug Administration) in marketbasket surveys indicates that the dietary dose used in the risk analysis may overestimate the actual dietary dose by more than 100 times (see p. F-90). To put worst case doses into perspective, the risk analysis estimates that the probability of a worst case dose occurring is 1.8×10^{-3} or 1 worst case dose for every 556 realistic doses (p. F-51).

The teratogenic potential of acephate is also discussed in Appendix F (p. F-93 to F-94). In studies where no effect was observed at any dose, the highest value tested is identified as the NOEL. All realistic acephate doses are hundreds to thousands of times below the NOELs listed in Table 1 for teratogenicity. The worst case doses for mixer/loaders, and for the observer and dietary exposure scenarios are more than 100 times below the NOEL for teratogenicity. It is significant to note that the lowest teratogenic NOEL value used in this comparison represents the highest dose tested with no effect observed. Furthermore, the next teratogenic NOEL value listed in Table 1 is 20 times higher than the lowest teratogenic NOEL, and is also the highest dose tested with no effect observed. This suggests that even the worst case doses identified in Appendix F are below teratogenic thresholds by a margin of safety that is greater than the safety factor used by EPA to establish the ADI.

The most severe impacts to human health are associated with doses that could be received from exposure to insecticide spills. Dermal exposures associated with aircraft spills are from 47 to 177 times below the highest exposure levels tested (with no effect) for the dermal LD_{50} (Appendix F, Table 12). It is estimated that up to 35 people could be exposed to such an accident. Fatalities are not likely to occur from short-term contact with acephate at these exposure levels; however, symptoms of cholinesterase inhibition are possible. Symptoms could include headache, dizziness, incordination, nausea, abdominal cramps, diarrhea, or sweating. Consumption of water into which aircraft spills have fallen result in realistic doses that are equivalent to the ADI, and worst case doses that slightly exceed the ADI. The reasons for suggesting that the worst case dose in water is probably still within an

acceptable margin of safety have been discussed earlier and in Appendix F (p. F-87 to F-90). Further discussion of potential health effects associated with aircraft spills needs to take into account the probability of the accident occurring.

Dermal exposures of acephate that are associated with tank truck spills result in dermal exposures that range up to twice as much as the highest exposure levels tested (without an effect) for the dermal LD_{50}. Spills in water result in possible oral doses that exceed the ADI by 10 to 18 times should 2 liters of water be consumed. It is not known what effects, if any, would result from dermal exposures to truck spills because no lethality was measured in the LD_{50} tests. However, cholinesterase inhibition would certainly occur. It is also probable that consumption of water into which acephate has spilled would cause symptoms of cholinesterase inhibition. As with aircraft spills, further discussion of potential health effects associated with truck spills needs to take into account the probability of the accident occurring.

The potential hazards associated with exposure to insecticide spills (both aircraft and vehicle) are real and have been identified. Fortunately, the probability of insecticide spills is extremely low. A low probability does not change the hazardous nature of the exposure, but rather estimates the likelihood that exposure to the hazard would occur. This provides the necessary information for the reader and decisionmakers to pass judgment on the acceptability of these risks.

As demonstrated in the risk analysis, the accident scenario associated with the greatest hazards to human health (truck spills) has the lowest probability of occurrence. For realistic doses the probability of occurrence is 1.08×10^{-5} on land or 1 spill for every 93,000 vehicle trips, and 1.2×10^{-6} in water or 1 spill for every 833,000 vehicle trips. For worst case doses the probability drops to 1.9×10^{-8} on land, or 1 worst case spill for every 50 million vehicle trips, and 2.2×10^{-9} in water, or 1 worst case spill for every 500 million vehicle trips. The probability of aircraft spills occurring for realistic doses are: 5.1×10^{-4} on land or 1 spill for every 1,960 aircraft loads, and 5.7×10^{-5} in water or 1 spill for every 17,554 aircraft loads. For worst case doses the probability of occurrence drops to 9.1×10^{-7} on land, or about 1 spill for every 1 million aircraft loads, and 1.0×10^{-9} in water, or 1 spill for every 100 million aircraft flights.

The risk analysis (Appendix F) indicates that all realistic doses and many worst case doses, with the exception of accidents, are either below the ADI or below NOEL values by

a margin of safety that is greater than 10 (the safety factor used by EPA to establish the ADI). Worst case doses are estimated to occur very infrequently (1 chance in 556). The risk analysis identifies accident scenarios where estimated doses may have an impact on human health. These are discussed, and the probability of occurrence identified so that decisionmakers and other readers can pass judgment on the acceptability of these risks.

The possible risk of cancer resulting from exposure to acephate applied to control gypsy moth are discussed in Appendix F. The maximum lifetime risk of cancer to an individual is 2.4×10^{-6}. In other words, about 300,000 acres would need to be treated with acephate in a single project before incident of cancer (above the number that would normally occur from other causes) would be observed in a population of over 4 million people. This level of cancer risk is in the same order (1 in a million risk) of magnitude as that associated with drinking 40 diet sodas or living two months in a brick or stone house (cancer caused by natural radiation).

As demonstrated in the analysis of human health risks presented in Appendix F, there is little risk of adverse human health effects for either the general public or occupationally exposed individuals as a result of using registered dose rates of acephate in gypsy moth suppression and eradication projects. Possible temporary effects related to cholinesterase inhibition are identified for sensitive populations based upon the worst case assumptions and exposure levels used in the analysis. These are a result of worst case exposure estimates for consumption of food and water that may contain residues of acephate. The only adverse health effects for workers or the general public identified are associated with major insecticide releases such as spills (probability of occurrence 10^{-4} to 10^{-6}), with a possible exposure to 2 to 3.5 people.

It is highly unlikely the registered use of acephate as applied to treatment areas during gypsy moth suppression or eradication project would pose any human health hazard.

Carbaryl

General Information. Carbaryl, trade name Sevin, is a broad spectrum organocarbamate used as a contact and stomach insecticide. In its technical grade, it is an odorless white to gray colored crystalline solid. Its melting point is 142°C, its density is 1.232 g/ml at 20/20°C, and its flammability is described by a Cleveland Open Cup Flashpoint of 193°C (Union Carbide 1968).

Since it was developed in 1956, carbaryl has become one of the most widely used insecticides. About 25 million pounds were used in the United States in 1974 (Dolinger and Fitch undated). Most of it was used in agriculture, but about 3.75 million pounds were used around houses and in gardens.

Such widespread use has prompted considerable investigation into effects which are now better understood than for most insecticides.

Fate in the Environment. Carbaryl is effective against members of most insect orders (Haynes et al. 1957; Barrett 1968). Insect species with more than 1 generation per year (USDA 1968) or with 1 generation with staggered development within the population often require repeated applications of carbaryl, because the chemical generally does not remain effective against the target insect for more than 1 or 2 weeks. The residue of carbaryl does have effective insecticidal property for several days after spraying. One study, showed that most saddled prominent larvae were killed within 48 hours of an application; however, larvae continued to die 8 days after spraying (Grimble et al. 1970). Skoog (1971) reported carbaryl effective at 18 days after treatment of grasshoppers. Residues of carbaryl, applied at 1 pound per acre as Sevin 4 Oil, remained high (causing 63 and 77 percent mortality in 2 groups of laboratory-reared gypsy moth larvae fed leaves from trees in a suppression area) at least 60 days after treatment. At 114 days mortality from the residues was 5 and 11 percent (Doane and Schaefer 1971).

Insecticide residues are degraded and diluted in the environment by a number of physical factors. For carbaryl, rain is a major factor in reducing residues (Union Carbide 1968). In Massachusetts, rain in excess of 1.8 inches occurred 12 to 24 hours after spraying with Sevin Sprayable, and the original 190 ppm residue of carbaryl or its degradation product on dominant scrub oak foliage was reduced to about 15 ppm 3 days after spraying (Wells 1966). Chemical decomposition on plants is less important, and plants absorb only small amounts (Union Carbide 1968). Once carbaryl is in soil or water, however, chemical decomposition is dominant and promptly leads to less toxic degradation products.

The half-life of carbaryl residues is 3 to 4 days. Carbaryl, in a Sevin 4 Oil formulation, was found to have a half-life of 8 to 10 days on range grasses (Fairchild 1970). On forest foliage, typical initial residues after treatment range from 30 to 100 ppm when carbaryl is applied for gypsy moth control. These decline to 5 to 20 ppm in 2 to 3 weeks (Back 1961). In Michigan, carbaryl (in that case, Sevin 4 Oil applied at a rate of 1 pound active ingredient per acre on maple trees showed residues of 500 ppm 1 day after spraying, 116 ppm after 8 days, 130 ppm after 15 days and 43 ppm after 35 days (Fairchild 1970). In New York, the same treatment applied to 2 mixed oak stands gave 192 and 55 ppm the day of spraying to 112 and 15 ppm 25 days after spraying (Fairchild 1970). Sampling of forest foliage may reveal excessively high or low residues in contrast to variation on row crops. This is believed due chiefly to the varied

terrain and air currents likely to be found over forested areas as opposed to agricultural crop land. Regardless of initial deposit, the rate of residue loss usually is constant.

In a monitoring study of a gypsy moth suppression project in which Sevin 4 Oil was applied at 1 pound per acre, exposed soil residues dropped below the detection limit (0.2 ppm) 128 days after spraying; the last samples showing residues had been taken at 64 days. Occasional samples of forest litter at 128 days still had slight residues (up to 0.65 ppm); but for most, residues had dropped below the limits of detection (Willcox 1972).

If carbaryl is applied unintentionally over open water such as small brooks or ponds, an initial deposit of 1 ppm or less in a water depth of about 4 inches may be expected to completely degrade or disappear in 1 or 2 days (Romine and Bussian 1971; Calif. Dep. Fish and Game 1963; Lichtenstein et al. 1966). Results were similar for water treated with Sevin 4 Oil during a gypsy moth suppression project (Willcox 1972). A proportionately lower concentration would occur in deeper water. More than 1 ppm in water is required to reach an LC_{50} value for fish. In a gypsy moth study, residues of 30 ppb in water dropped to 1.5 ppb in 1 day (USDA 1964).

Karinen et al. (1967) concluded that carbaryl reaching shallow mud flats in marine ecosystems probably would be rapidly removed from water by adsorption on bottom mud. Chemical degradation then occurs, with carbaryl and 1-naphthol likely to persist in mud for 2 to 6 weeks. Carbaryl as an 80 percent wettable powder was applied at 10 pounds per acre to a mud flat at low tide, simulating application for control of oyster pests. The initial residue (10.7 ppm) dropped rapidly the first day when the tide removed carbaryl and 1-naphthol not adsorbed on mud. The toxicant in the top inch of mud declined from 3.8 ppm to 0.1 ppm 42 days later.

Carbaryl decomposes or metabolizes to several substances, of which 1-naphthol and 1-naphthyl (hydroxymethyl) carbamate are the most important (Union Carbide 1969). The relative toxicities (LD_{50}) of carbaryl and these substances to male rats are: carbaryl, 500 mg/kg; 1-naphthol, 2,590 mg/kg; and 1-naphthyl (hydroxymethyl) carbamate, more than 5,000 mg/kg. The no-ill-effect levels over a 7-day period for the same 3 substances are 125 to 250 mg/kg, 500 to 1,000 mg/kg and 250 to 500 mg/kg, respectively.

Toxicology. Application of carbaryl for gypsy moth control is likely to adversely affect some beneficial insects. However, any reduction in nontarget insects that may occur as a result of carbaryl application is temporary (Karpel 1973; Moulding 1972). Johansen (1959) reported carbaryl as highly toxic to honeybees, though different formulations of

carbaryl have different levels of toxicity. The difference in toxicity is due mainly to the manner in which these formulations dry on the target foliage, which, in turn, determines how readily the insecticide can be picked up by honeybees and transported to the hive. Apiaries can be protected by taking precautionary measures such as locating hives beyond bee-flight range until 1 week after application (Strang et al. 1968). Covering hives before treatment also can reduce losses (Jaycox 1963).

The registered use of carbaryl has no direct adverse effects on amphibians or reptiles or fish (Romine and Bussian 1971, Tompkins 1966; Willcox 1972; Pillow 1973).

During operational spraying in Maine (1.0 lb carbaryl/acre), acetylcholinesterase levels were depressed an average of 20 percent in brook trout (Salvelinus fontinalis Mitchill) and 35 percent in Atlantic salmon (Salmo salar C.) (Hulbert 1978). These depressions were detected within 24 hours of spraying and persisted throughout the sampling period (192 hours). During spruce budworm spraying in Maine (1.0 lb carbaryl/acre) in 1975, brook trout depressions ranged between 13 and 22 percent and returned to normal within 48 hours. Activity depressions in Atlantic salmon were more gradual (9 to 27 percent) and failed to return to normal within the same sample period (48 hours) (Marancik 1976).

A study of buffered streams by McCullough and Stanley (1980) during the 1979 Maine spruce budworm project indicated that feeding and acetylcholinesterase activity of young-of-the-year brook trout were not adversely affected. Ott et al. (1980) studied the effects of an application of 0.75 lb carbaryl/acre on young brook trout in 1 unbuffered and 1 buffered stream in Maine. No physiological changes in brook trout were detected that could be attributed to carbaryl contamination. In addition, these workers concluded that it was extremely unlikely that streams accidentally contaminated by carbaryl during spraying for spruce budworm control in Maine would have resulted in fish mortality.

Some aquatic insects in the orders Plecoptera (stoneflies) and Ephemeroptera (mayflies) are known to be highly sensitive to low levels of carbaryl. Trichoptera (caddisflies) and Diptera (true flies) also are sensitive to carbaryl. There may be a 50 to 100 percent reduction in aquatic insect populations in treated streams and ponds (Burdick et al. 1960). LOTEL (1977) reported that in a stream treated with 1.0 lb carbaryl/acre, each sampling station recorded a residue of at least 40 ppb and a peak residue to 80 ppb. The biological impact was indicated by increased drift of dead and dying stoneflies, mayflies, caddisflies, and true flies.

The effects of 2 consecutive years of spraying on other aquatic organisms appear similar to those observed in areas treated just once (Trial 1978, 1979; Courtemanch and Gibbs 1978). These effects include loss of stonefly species from individual streams, and altered generic assemblages for an indefinite period (Trial 1978, 1979). A study of buffered streams by McCullough and Stanley (1980) during the 1979 Maine spruce budworm spray project indicated that benthic invertebrate fauna were not adversely affected. Also, the numbers of drifting invertebrates were substantially lower than in previous years. The amount of long-term impact appears to be a function of species susceptibility and recolonization ability. Two consecutive years of spraying with carbaryl reduced populations of stonefly and susceptible mayfly genera to near zero.

Carbaryl lowers the cholinesterase levels in many animals. Cholinesterase splits acetylcholine, the chemical responsible for forming the bond necessary to carry an impulse through the nervous system. If the acetylcholine is not split, the impulse is repeated again and again, and a severe lowering of cholinesterase will result in symptoms of nerve poisoning.

The acute oral LD_{50} is 500 to 850 mg per kilogram of body weight (mg/kg) (Meister 1983). No observable effect levels (NOELs) have been established in laboratory animals for a variety of effects (Appendix F, Table 2). The lowest established NOEL is 3.25 mg/kg/day in pregnant dogs for teratogenic effects. Other established NOEL's for teratogenic effects in mice, rabbits, and rats range from 150 to 500 mg/kg/day.

Possible dermal and oral exposures to domestic and wild animals from the use of carbaryl in gypsy moth projects are evaluated in Appendix F (p. F-32 through F-36). Goats and rabbits were used as surrogates for large (goat) and small (rabbit) animals. Dermal doses for large animals were estimated to range from a realistic value of 0.02 mg/kg/day to a worst case value of 0.2 mg/kg/day. Oral doses could range from 0.29 mg/kg/day (realistic) to 2.88 mg/kg/day (worst case). Doses to small animals were slightly larger because of the greater surface-area to body weight ratio. Dermal doses could range from 0.04 mg/kg/day (realistic) to 0.4 mg/kg/day (worst case). Oral doses could range from 0.30 mg/kg/day (realistic) to 3.00 mg/kg/day (worst case). Total exposure to any animal would be the sum of the dermal and oral doses, for example, total dose to a large animal would be 3.08 mg/kg/day (worst case).

These dermal and oral exposures can be compared to the acute oral LD_{50}, acute dermal LD_{50}, and NOEL values summarized in Tables 2 and 7 in Appendix F for various animals. The estimated worst case doses to animals are orders of magnitude below doses that cause effects in test animals,

with the possible exception of teratogenicity in dogs. The teratogenic threshold for dogs is only 6.25 mg/kg/day (Table 2 in Appendix F) which is close to the estimated worst case dose to small animals of 3.4 mg/kg/day. The probability of the worst case dose occurring is one chance in 556 (Appendix F, F-51), which would approximate the probability that a pregnant dog might be exposed to carbaryl at the worst case dose and then bear deformed pups.

Depression of cholinesterase activities has, however, been reported in birds. Depressed brain acetycholinesterase activity of forest birds was reported following application of 1.0 lb carbaryl per acre in Montana (Zinkl et al. 1977), while split treatments (0.31 and 0.69 lb carbaryl per acre) in Maine revealed no depression (Gramlich 1979). Observations by Connor (1960) on 49 species of birds exposed to carbaryl failed to reveal adverse effects on their behavior, conditions, or reproduction and rearing of young.

In a study of the response of breeding birds to an aerial application of carbaryl, Zinkl et al. (1979) reported no significant effects on the numbers of breeding birds, nesting success, mortality rates, or activities of brain cholinesterase. An indirect effect of carbaryl spraying to birds may be a depletion of available food, which alters bird activity (Doane and Schaefer 1971).

Harry (1977) compiled an extensive review of human exposure to carbaryl. Despite almost universal exposure in the United States over more than 20 years, it seems that the safety record of carbaryl is almost unparalleled by any other insecticide.

In forest openings, actual dermal exposure studies conducted by the South Carolina Epidemiologic Studies Center (1979) during Maine's spruce budworm spray project showed a total dermal exposure of 10 mg carbaryl for a person (150 pounds) who is 80 percent clothed at the time of application.

In this study, the person respiratorily exposed for 2 hours in the spray area would receive only 0.054 percent carbaryl of the Time Weighted Average (TWA) standards. This equals a safety margin of 1,834 times the occupational exposure (personal communication, Ernest Richardson, Maine Bureau of Health).

In 1978 and 1979, field studies were conducted by the South Carolina Epidemiologic Studies Center to measure human exposure to carbaryl during spruce budworm suppression projects in Maine. The level of carbaryl residues found in urine samples taken during the 1978 project are shown in Table 3. The following quotations regarding 1978 results are taken from the Draft Interim Report (SCESC 1978):

Table 3. 1-Naphthol residues in urine samples from persons exposed to Sevin 4 oil in 1978. 1/ 2/

Exposure group	Number of tested participants	Number of urine specimens tested	Number tested positive	Range of positive tests (ppb)	Median residue level of positive tests (ppb)	Average residue level of positive tests (ppb)
Pilots	5	10	10	41.00-1750.00	121.50	323.89
Loaders	5	9	8	83.00-5540.00	656.00	144.21
Ecologists	9	17	8	14.00-146.00	28.66	51.87
Wardens/ rangers/wives	11	11	5	11.11-25.00	12.14	16.85
Scouts	10	10	4	10.77-23.00	14.58	15.73
Lab technician	11	11	3	11.25-16.25	13.68	13.73
Residents	42	50	7	10.00-37.60	14.00	15.63
EPA/safety	5	6	5	23.00-1250.00	89.14	313.99

1/ Urine 1-naphthol residue analysis; lowest level detectable by this method is 10 parts per billion (ppb).

2/ From Draft 1978 Interim Report, Measurement of Exposure to the Carbamate Carbaryl: Maine Carbaryl Study, 1978. South Carolina Epidemiologic Studies Center, Medical University of South Carolina, March 1979. (Used by permission of EPA.)

Table 4. 1-Naphthol residues in urine samples from persons exposed to Sevin 4 oil in 1979. 1/ 2/

Exposure group	Number of tested participants	Number of urine specimens tested	Number tested positive	Range of positive tests (ppb)	Median residue level of positive tests (ppb)	Average residue level of positive tests (ppb)
Pilots	2	2	1	156.87		156.87
Ecologists	3	3	1	25.75		25.75
Scouts	6	6	4	10.42.17.90	12.80	13.48
Ranger/family	6	6	2	29.11-62.46	45.78	45.78
Field technician	7	7	2	14.23-187.48	100.86	100.86
Residents	16	16	5	24.00-2556.0 3/	199.40	615.97
Safety	1	1	1	3029.00 4/		3029.00

1/ Urine 1-Naphthol residue analysis; lowest level detectable by this method is 10 parts per billion (ppb).

2/ From Draft Interim Report, Measurement of Exposure to the Carbamate Carbaryl: Maine Carbaryl Study, 1979. South Carolina Epidemiologic Studies Center, Medical University of South Carolina, November 1979. (Used by permission of EPA.)

3/ The 2556.00 figure is probably due to the use of a home garden dust containing carbaryl.

4/ Twelve to 24 hours after a second intense dermal exposure.

> Human exposure to carbaryl applications during Maine's 1978 Spruce Budworm Suppression Project was monitored by the South Carolina Epidemiologic Studies Center. Except where there had been exposure to carbaryl from either mixing operations or home usage, none of the urine samples collected prior to application were found to contain the alpha-naphthol metabolite. Analyses of urine samples collected 12 to 24 hours after application found that the cohorts of pilots and aircraft loaders had the highest residues. About one-half of the samples from ecologists and rangers who were working and/or living in the application areas showed small but measurable levels of alpha-naphthol. Of the 49 urine samples collected from residents 12 to 24 hours after application, only 7 were positive for alpha-naphthol and ranged from 14 to 38 ppb. From the administration of health effect questionnaires, it was determined that no participant reported symptomatology thought to be related to carbaryl exposure.
>
> Data presented in the draft 1978 Maine Carbaryl Study report suggest that there were no apparent risks to those workers occupationally exposed to and individuals residing near areas aerially treated with carbaryl. Alpha-naphthol residues in the residential participants indicated that drift did not occur.

Because of continued public concern and the need to further investigate the amount of human exposure that results from an aerial application of carbaryl, a study was conducted in 1979 by the South Carolina Epidemiology Studies Center to monitor the exposure of humans to carbaryl by measuring the urinary metabolite, alpha-naphthol, in persons potentially exposed during the aerial application to forests and to relate this exposure to air sampling. Results of 1979 urine residue analyses are shown in Table 4. The following quote regarding results of this work is from their draft interim report (SCESC 1979):

> The National Institute of Occupational Safety and Health (NIOSH) has established a time weighted average (TWA) for occupational exposure to carbaryl. The TWA is a maximum exposure limit for occupational exposed employees based on a 10-hour work shift, 5 mg/m^3. The TWA, when compared to the air sampling results of the Washburn area, indicates that the residents located 0.6 miles north of spray block 6-14 were exposed to carbaryl concentrations in the magnitude of thousandths of one percent of the permissable occupational level. The highest reported level of carbaryl equivalent was found at Site 1 during the first 12 hours of sampling after application. This level (341.0 ng/m^3) when converted to milligrams equals 0.0003 mg/m^3 or 0.006 percent of the TWA standard. Thus the exposure of residents to carbaryl concentrations in environmental

air throughout the sample period was the smallest fraction of allowable levels mandated for more intensive occupational exposure.

In the 1979 study, individuals who remained indoors during a nearby application of carbaryl were found to have no detectable alpha-naphthol, a metabolite of carbaryl, in their urine with the exception of one person who may have been previously exposed to another source of carbaryl or the insecticide malathion. Persons outdoors at the same location were found to have detectable levels. The same study indicated that persons entering spray blocks more than 24 hours after carbaryl application probably would have a 5 percent or less chance of receiving detectable exposure to carbaryl (personal communication, Dr. Sandifer, South Carolina Epidemiologic Studies Center).

In 1978 the New Jersey Department of Health, Epidemiologic Studies Program-Pesticides, monitored people residing in gypsy moth treatment areas. The study site consisted of approximately 23 acres of heavily wooded residential land containing approximately 80 dwellings. Following carbaryl application, researchers were unable to detect the presence of a metabolic indicator of carbaryl in the urine of homeowners residing in the treatment area. By contrast, pesticide mixing and loading personnel exhibited levels of the indicator metabolite. However, a study of carbaryl formulators, conducted by the New Jersey Epidemiologic Studies Program during 1967-73, found no relationship between excessive and long-term exposure to carbaryl and chronic adverse health effects (NJESP 1974). The 1978 study further suggested that individuals who remain indoors during insecticide application receive no measurable exposure to the material. The report, presenting the 1978 study results as submitted to EPA, concluded that the aerial application of carbaryl to communities as conducted resulted in no measurable threat to human health (Schulze et al. 1979).

Results from the studies in Maine (SCESC 1978, 1979) and New Jersey (Schulze et al. 1979) indicate that, while precautions can be taken to reduce the number of people exposed and the amount to which they are exposed, it is not possible to avoid exposing some people to carbaryl during the spray operation. However, the amount of carbaryl is extremely small and exposure can be further minimized by remaining indoors or outside of the treatment areas during application.

Acute toxicity to people is rarely a problem with carbaryl. Comer et al. (1975) reported that plant workers producing carbaryl are exposed dermally to average levels of 73.9 mg/hr and respiratorily to 1.1 mg/hr of work. Urine samples of plant workers had concentrations of 8.9 ppm 1-naphthol, a metabolite of carbaryl, with average excretion rates of 0.5 mg/hr. In the same study, the exposure levels of spray

applicator workers were studied. Average carbaryl levels were 59 mg/hr dermally and 0.09 mg/hr respiratorily. Comer et al. (1975) concluded that at these dose levels, concerns about acute toxic effects are minimal. Controlled human studies with carbaryl have been conducted at dosages sufficient to cause significant adverse effects. One investigation showed that a daily administration of carbaryl to human volunteers at 0.06 and 0.13 mg/kg/day for 6 weeks caused only slight signs and symptoms attributable to the insecticide (Wills et al. 1969).

Carbaryl is not a chronic poison. Test animals can tolerate a substantial percentage of an acutely toxic dose in the diet daily for a lifetime. Levels causing no significant effect are as high as 400 ppm dietary to the mouse, equal to 60 mg/kg body weight daily, or 200 ppm to the rat, equal to 10 mg/kg (personal communication, R.C. Back, Union Carbide Agricultural Products Company). These levels are well below the estimated maximum exposures from realistic and worst case doses associated with gypsy moth suppression and eradication projects (Appendix F).

There is some evidence suggesting that carbaryl has teratogenic potential (causes birth defects). Smalley et al (1968) reported a low irregular incidence of birth defects in the offspring of beagle dogs given doses as low as 6 mg/kg per day in the diet. Evidence of teratogenicity was observed in offspring of rabbits given 150 or 200 mg carbaryl/kg per day by gavage. In both cases, some maternal toxicity was observed, making it uncertain whether the effects were due to the carbaryl or whether they were mediated through maternal toxicity (Murray et al. 1979). Although a dose (given by gavage) of 150 mg carbaryl/kg/day instituted signs of colinesterase inhibition and maternal toxicity in adult mice, daily doses of 1166 mg/kg/day given in diet resulted only in decreased maternal body weight gain. Other studies report no teratogenicity in guinea pigs given 200 or 300 mg carbaryl/kg/day (Weil et al, 1973) or in hamsters given a dose that caused maternal toxicity (Robens 1969). The low teratogenic NOEL in dogs compared to other test animals has raised a question in the scientific community. EPA has recently issued registration standards for carbaryl and has requested the registrants to repeat the teratology in beagle dogs (EPA 1984a). In gypsy moth projects, the exposure levels, realistic and worst case, estimated for the general public, sensitive populations, and occupationally exposed individuals are well below the lowest established NOEL for teratogenic effects (Appendix F, Tables 9 and 14).

The question of potential teratogenic effects in humans is addressed in a letter dated May 16, 1979, from Mr. Douglas D. Campt, Director of Registration Division, EPA, to Mr. William M. Cranston (now retired), N.J. Department of Agriculture. The letter includes the following statement:

Since experimental exposure to carbaryl has caused birth defects in dogs, carbaryl may have some potential to do so in humans, and the Environmental Protection Agency is currently attempting to assess that potential. However, since a teratogenic study of carbaryl in rhesus monkeys was negative, it would appear that the teratogenic potential in humans, if any, is not great. One can never conclude that risk from exposure to any chemical is zero, and it is only reasonable and prudent to suggest that women who may be pregnant should avoid any unnecessary exposure to carbaryl and other chemicals. This is easily accomplished in the use of carbaryl by remaining indoors or under suitable cover at the time the application is made. Once the spray settles, any further potential for exposure is greatly reduced, and should be of no concern.

Possible realistic and worst case exposure levels to the general public or workers are analyzed in Appendix F. Workers in the mixer/loader group have the highest potential exposure with doses ranging from 0.046 mg/kg/day (realistic) to 0.20 mg/kg/day (worst case). Exposure to the general public included possible direct exposure to the insecticide, and possible indirect exposure from touching insecticide residues on grass, toys, cars, etc., or eating food that may contain carbaryl residues. Greatest possible exposure would occur to individuals who are outside (observers) and receive a direct application and then consume only food and water containing residues of carbaryl. The estimated doses to this highest exposure group (designated observer and dietary in Table 9, Appendix F) range from 0.012 mg/kg/day (realistic) to 0.174 mg/kg/day (worst case).

When the probable exposures to workers and the general public were compared to the ADI (Acceptable Daily Intake) set by the U.S. Environmental Protection Agency or NOELs (determine from animal studies), all realistic doses were found to be below the ADI and are therefore considered to be within acceptable margins of safety. The worst case dose that includes possible exposure from eating and drinking contaminated food and water exceed the ADI. However, even these worst case doses were found to be below the teratogenic threshold (NOEL) for most animals (excluding dogs) by margins of safety greater than 100 (see page F-89 to F-90 in Appendix F). Furthermore, as was discussed earlier in the acephate section, the dietary doses used in the risk analysis are more than 100 times greater than actual carbaryl residues found on foodstuffs, according to FDA marketbasket surveys. This strongly suggests that the worst case doses that include a dietary component overestimates risks.

Dermal exposure and possible water contamination that could result from some accidents are summarized in Appendix F, Table 13. An aircraft spill, which could affect a total of 35 people, could result in short term acute dermal exposures ranging from 257 mg/kg (realistic) to 468 mg/kg (worst case). Even the worst case dose is below the dermal LD_{50} value established from animal studies by margins of safety greater than 100. Any human exposure resulting from drinking water contaminated by a carbaryl spill would be about the same as the ADI established by EPA. Acute dermal exposures resulting from truck spills (27000 mg/kg) pose a potential hazard to individuals involved with the spill. These high acute exposures exceed the dermal LD_{50} value by about a factor of 3. Exposure associated with drinking water contaminated by truck spills should also be considered hazardous because the level (0.604 mg/kg) exceeds the ADI. Although doses resulting from truck spills pose potential hazards to individuals involved with them, the probability of occurrence is very low (1.08×10^{-5}), about one chance in 100,000. The probability of aircraft spills is somewhat higher (5.7×10^{-4}), or less than one aircraft spill for every 1000 trips flown.

Halpin (1980) investigated the possibility of increased birth defects (teratogenicity) in New Jersey municipalities where carbaryl was aerially applied for gypsy moth suppression and whether or not a relationship in time between the occurrence of birth defects and this spraying existed. Although this was not an exhaustive study of birth defects in the 3-county area examined, it did provide a basis for concluding that there was no association between the spraying of carbaryl (Sevin 4 Oil) for gypsy moth and the birth defects reported from Cape May County.

The carrying agent and emulsions of the Sevin 4 Oil formulation, as with other insecticides, are a trade secret. However, investigations have shown that the formulation contains no significant quantities of polynuclear aromatics which are compounds suspected of being carcinogenic. Nonionic polymers of polyoxyethylene ethers and nonyl phenol substances, which have been implicated in Reye's Syndrome, are not present.

The question of viral potentiation of Sevin 4 Oil recently was studied by two University of Maine researchers. Their data suggest that viral potentiation may be associated with exposure to Sevin 4 Oil. The Maine Bureau of Forestry appointed a panel of medical experts to review this study and to make recommendations concerning the potential health effects of Sevin 4 Oil. They concluded that Sevin 4 Oil poses a "potential but inconclusive health risk" and

recommended that the Maine Bureau of Forestry develop more stringent limitations so that "no uninformed or unconsented human exposure will occur during a forest spray operation." A followup study was undertaken to determine the component of the Sevin 4 Oil constituents that may be viral enhancing. The data indicate that the active ingredient, carbaryl, is responsible for the viral enhancement. The medical advisory panel reviewed these new findings and felt that the original recommendations were still valid.

Viral enhancement has only been demonstrated in laboratory tissue culture. Tests in 26 species of animals have not demonstrated any carbaryl-induced viral enhancement (statement by Antoine Puech, Union Carbide, Salem, Oregon, Gypsy Moth Public Hearing Record, 1982).

Under laboratory conditions, carbaryl has been reacted with nitrite compounds in the presence of an acid catalyst and heat, to form N-nitrosocarbaryl. This laboratory synthesized N-nitrosocarbaryl has been used in several laboratory test systems to demonstrate its potential mutagenic properties. Such diverse test systems as microorganism bioassays, cell cultures, bone marrow, and transplacental host-mediated trials have been conducted (Uchiyama et al. 1975; Elespuru and Lijinsky 1973; Siebert and Eisenbrand 1974). Stomach cancer and local sarcomas have been produced in rats when laboratory-synthesized N-nitrosocarbaryl was used in feeding studies or when subcutaneously injected (Eisenbrand et al. 1975; Lijinsky and Taylor 1976). However, repeated dermal applications failed to produce skin tumors in the same species.

Since repeated dermal exposures did not produce skin tumors, oral exposure was investigated. It is thought that oral exposure to N-nitrosocarbaryl occurs by carbaryl (in the form of residues) and sodium nitrite (in saliva or food) combining in the stomach under acid conditions. In studies with guinea pigs, the formation of N-nitrosocarbaryl was reported when sodium nitrite and carbaryl were present in the stomach (Rickard et al. 1979). However, the *in vivo* production of N-nitrosocarbaryl was less than 0.2 percent of that obtained from the *in vitro* production. Further, the low pH of the guinea pig stomach, which is similar to the human stomach, causes the N-nitrosocarbaryl to become rapidly denitrosated to form carbaryl. In other laboratory feeding studies, high levels of physically mixed nitrite and carbaryl did not produce a significant increase in tumors or other lesions in either pregnant or nonpregnant rats or the exposed progeny (Lijinsky and Taylor 1977). Other laboratory studies were conducted with rats and mice to determine the oncogenic potential of carbaryl.

Significantly, these studies did not produce oncogenicity attributable to carbaryl even though many were conducted at or near the maximum tolerated dose for up to 2 years. N-nitrosocarbaryl can cause mutagenic and carcinogenic effects. When found in the living body, it is unstable and the quantity is insufficient to cause carcinomas as demonstrated by these studies. The EPA review of the N-nitrosocarbaryl issue is presented in Appendix C.

The possible risks of cancer resulting from N-nitrosocarbaryl are discussed in Appendix F by making the worst case assumption that carbaryl would be converted to N-nitrosocarbaryl in the stomach (p. F-94 to F-96 in Appendix F). The worst case lifetime risks of cancer to individuals exposed to carbaryl (and thus N-nitrosocarbaryl) is 2.79×10^{-9}. This level of cancer risk is more than 100 times lower than the risks of cancer caused by smoking 2 cigarettes, drinking 40 diet sodas, or eating 90 lb of charcoal broiled steaks in a lifetime.

The overall conclusion in the risk analysis (Appendix F) is that even worst case exposures that might result from the use of carbaryl to control gypsy moth are below threshold doses for specific health responses (e.g. birth defects) by margins of safety greater than 100. Risk of cancer or heritable mutations is below the level of risk that society apparently accepts (1×10^{-6}). Based on a review of mutagenicity studies, carbaryl was found to pose a low risk for heritable mutations in humans.

Following an extensive review of available studies relating to the insecticide carbaryl, the EPA has concluded that further restrictions of pesticide products containing carbaryl were not warranted. A summary of that decision is presented in Appendix D of this FEIS. In view of the existing data and the results of the analysis of human health risks presented in Appendix F, it is highly unlikely that the registered use of carbaryl, as applied to treatment areas during gypsy moth suppression or eradication projects would pose a human health hazard.

Diflubenzuron

General Information. Diflubenzuron, trade name Dimilin, acts as an insect growth regulator by interfering with the synthesis of chitin, a polyglucosamine found in the body wall of insects. The primary effect is by ingestion, but there is minimal contact action. Diflubenzuron slowly acts during the gypsy moth larval stage causing the body wall of the insect to rupture during the molting phase. The current EPA label interpretation restricts the use of Dimilin 25W --the formulation of diflubenzuron used for gypsy moth control--to forested areas with 1 house or less per 10 acres. Diflubenzuron also is registered for control of cotton boll weevil, several insects on soybeans, and mosquito larvae.

It is a white crystalline solid almost insoluble in water (about 0.2 ppm) and apolar solvents. In most polar to very polar solvents the solubility is moderate to good.

Fate in Environment. Diflubenzuron is rapidly degraded (3 to 4 days) in soil. The degradation was unrelated to soil type but was very much dependent on both the microbial activity in the soil and the particle size of the diflubenzuron (Willcox and Coffey 1978). Studies at Brigham Young University (Pintar et al. 1975) showed that all soil bacteria could utilize diflubenzuron as a sole carbon or sole carbon and nitrogen source.

The persistence of diflubenzuron in water and soil-water systems is, as with soil alone, related to the microbial activity and the particle size of the material applied. With agricultural soils, the half-life in hydrosoils is 0.5 to 1.0 weeks for the parent compound and 8 weeks for the entire radiocarbon residue (Willcox and Coffey 1978).

Toxicology. Studies have been conducted on the effects of diflubenzuron on a number of nontarget species in the forest ecosystem (USDA 1975; Willcox and Coffey 1978).

In these studies, several different forest ecosystems were treated with diflubenzuron at rates from 0.03 to 0.06 lb active ingredient per acre. Following application, soil microbes and invertebrates, terrestrial insects, aquatic insects and other nontarget crustaceans, fish, small forest mammals, and birds were monitored for the effects of treatment. No treatment-related effects were observed with elements of the soil community, including soil microbes and fungi, soil inhabiting mites, and collembolans. It was shown that diflubenzuron at the rates applied had no effect on the organisms that are involved in the degradation and use of the forest leaf litter. In the studies of terrestrial insects, the single application of diflubenzuron had no effect on the free-flying, forest-inhabiting insects. Honeybees were unaffected when hives were placed directly within test areas. The effects monitored included honey production, egg production by the queen, and brood hatch development and survival (Willcox and Coffey 1978). Even though potential exposure to insectivorous small mammals and birds was possible, no treatment related effects were observed. Species composition and territorial distribution remained unchanged (Willcox and Coffey 1978).

Other studies have been conducted in aquatic habitats to determine the effect of diflubenzuron on aquatic insects and nontarget crustaceans (Mulla et al. 1975; Steelman et al. 1975; and Miura et al. 1975). Diflubenzuron has been found to reduce populations of certain sensitive nontarget crustaceans, primarily water fleas, cyclops and immature copepods, and certain species of aquatic insects (mayflies, corixids, and notonectids).

The effect on the aquatic environment is extremely variable and, although the species diversity in this habitat often is altered, populations of the nonsensitive forms adjust the overall community numbers to counteract the effects. Therefore, the limited environmental impact due to the nonpersistence of diflubenzuron is short lived and population recovery of the more sensitive species occurs within 14 to 28 days in most cases (Willcox and Coffey 1978).

The acute toxicity of diflubenzuron to mammals has been investigated by Phillips-Duphar B.V., Harris Laboratories, and the Huntingdon Research Center (Willcox and Coffey 1978). Because of its mode of action, the interruption of chitin synthesis on the insect, diflubenzuron has low mammalian toxicity. Diflubenzuron (40 mg technical) was shown to be a marginal eye irritant, but 50 mg in an aqueous gum tragacanth solution was not irritating. When diflubenzuron was tested for dermal effects, it was found to be nonirritating. The very low toxicity of diflubenzuron for mammalian and nonmammalian species exclusive of insects and certain chitin containing arthropods is in part related to the ability of the compound to be absorbed by the animal exposed and its ability to biochemically detoxify and eliminate diflubenzuron from its system (Willcox and Coffey 1978).

The acute oral LD_{50} in laboratory animals for diflubenzuron is greater than 4,640 mg per kilogram of body weight (mg/kg) (Meister 1983). No observable effect levels (NOELs) have been established in laboratory animals for a variety of effects (Appendix F, Table 3). These range from 1.1 mg/kg/day (no effect) for an 80-week feeding study in mice to 8 mg/kg/day (highest dose tested) for a 3 generation feeding study in mice. In both cases the only adverse effect observed was elevated methemoglobin and sulfhemoglobin levels. No other effects of any kind were observed. NOEL values for teratogenicity (birth defects) are set at 4000 mg/kg/day (highest dose tested).

The diflubenzuron exposure levels associated with gypsy moth suppression and eradication projects have been estimated for animals (Appendix F, p. F-32 to F-36). For large animals the realistic dose is estimated to be 0.019 mg/kg/day [(0.02 mg/kg/day + 0.29 mg/kg/day) x 0.06]. The worst case dose is estimated to be 0.18 mg/kg/day [(0.2 mg/kg/day + 2.88 mg/kg/day) x 0.06]. For small animals the realistic dose is estimated to be 0.02 mg/kg/day [(0.04 mg/kg/day + 0.3 mg/kg/day) x 0.06]. The worst case dose is estimated to be 0.20 mg/kg/day [(0.4 mg/kg/day + 3.0 mg/kg/day) x 0.06]. Realistic and worst case doses for large and small animals are 10 to over 100 times less than the lowest NOEL value,

and greater than 20,000 times below the NOEL value for teratogenic effect. It is highly unlikely that animals, large or small, wild, domestic or livestock, would be adversely affected at the realistic exposure levels.

The risk analysis (Appendix F, Table 10) indicates that all realistic and worst case doses to the general public and the occupationally exposed group are equal to or below the ADI established by EPA. Previous discussions concerning the conservative nature of the dose estimate for the dietary component can also be made here (see acephate section, and Appendix F, p. F-90). The doses and exposures (realistic and worst case) associated with aircraft and tank truck spills are all below the established ADI and below the highest exposure level tested (with no effect) for the dermal LD_{50}. The probability of such accidents occurring are the same as those discussed previously for acephate.

The risk analysis demonstrates that all doses (realistic and worst case) associated with the use of diflubenzuron are equal to or below the established ADI. This suggests that these doses are well within an acceptable margin of safety. The ability of diflubenzuron to cause elevated methemoglobin and sulfhemoglobin levels has caused concern that exposure might result in impaired oxygen transport. EPA has set a NOEL of 1.1 mg/kg/day for this effect in mice. Estimated worst case diflubenzuron doses to workers or residents are at least 92 times below that NOEL. Finally, on the specific issues of mutagenicity and cancer, diflubenzuron was found to be nonmutagenic even at high dose levels (Appendix F, p. F-12) and noncarcinogenic in oncogenicity studies recently reviewed by EPA (Appendix F, p. F-14). Even though diflubenzuron itself is noncarcinogenic, there is uncertainty about the carcinogenic potential of one of its metabolites, 4-chloroaniline. The cancer risk associated with eating fish or meat containing this metabolite as a result of diflubenzuron spraying is discussed in Appendix F. Worst case lifetime risks to an individual were estimated to be in the 1×10^{-8} to 1×10^{-9} range. This risk level is 100 to 1000 times below the one in a million (1×10^{-6}) cancer risk associated with smoking 2 cigarettes in a lifetime or eating 6 pounds of peanut butter in a lifetime.

Trichlorfon

General Information. Trichlorfon, most commonly known as Dylox , is an organophosphate chemical that is used as an insecticide and as a therapeutic drug to treat selected endoparasites in humans and livestock (Abdalla et al. 1965; Beheyt et al. 1961; Davis and Bailey 1969; Wegner 1970). Trichlorfon also is registered for use on beef and dairy cattle for the control of ectoparasites (EPA 1969). The insecticide trichlorfon is registered for use on a variety of field crops, vegetables, seed crops and ornamentals. It is effective for control of many different species of insects with contact and ingestion modes of action.

Technical trichlorfon is a white crystalline solid with a specific gravity of 1.73 at 20.4°C. Solubility is 12 percent in water at 26°C and it is soluble in alcohols and ketones.

Fate in Environment. Trichlorfon is rapidly degraded in the environment. In New York (Judd et al. 1972), trichlorfon was found in small amounts in water samples collected immediately after spraying, but the concentration of the chemical dropped below a detectable level 4 days after spraying; the half-life of trichlorfon in water at 30°C was 4.7, 0.6, and 0.1 days at pH levels of 5, 7, and 9, respectively. In this test, water was protected from light. In an outdoor pond (pH 7.0) at temperature 20°C, and with exposure to sunlight and wind, trichlorfon showed a half-life of only 0.3 day (Chemagro 1971).

Doane and Schaefer (1971) found that gypsy moth larvae that were fed leaves collected 12 days after treatment experienced only 2.5 percent mortality. After an application of 1.0 lb trichlorfon per acre in New York for gypsy moth, Weiss et al. (1973) reported that residual levels dropped sharply within a few days after treatment, and by 60 days had reached the following percentages of their initial levels: 15 in leaves, 5 in litter, 10 in unexposed soil, and less than 1 in exposed soil.

Toxicology. Trichlorfon has shown no significant adverse effects against vertebrates, birds, reptiles, amphibians, and fish (Lewallen and Wilder 1962; Pearce 1970; Chambers 1972; Caslick and Smith 1973; Finger and Werner 1973; and Todaro and Brezner 1973). Bird activity may be temporarily altered through the reduction of insects available for food (Doane and Schaefer 1971; Caslick and Cutright 1973). Trichlorfon is classified as having a low toxicity for bees (Johansen 1959). Trichlorfon residues are not transported by foraging bees from contaminated surface into hives (Gilpatrick and Terrill 1970).

When used in accordance with the label, trichlorfon applied at dosages used for gypsy moth treatment will reduce populations of some nontarget insects, including some parasites and invertebrate predators. These nontarget insect populations recover, some within a few weeks (Chemagro 1968).

Possible dermal and oral exposure to domestic and wild animals resulting from the use of trichlorfon to control gypsy moth are estimated in Appendix F (p. F-32 to F-36). Dermal doses to large animals (goat as the surrogate) range from 0.02 (realistic) to 0.20 mg/kg/day (worst case). Oral doses to large animals could range from 0.29 (realistic) to

2.88 mg/kg/day (worst case). Dermal doses to small animals (rabbit as the surrogate) ranged from 0.04 (realistic) to 0.40 mg/kg/day (worst case); oral doses ranged from 0.30 (realistic) to 3.00 mg/kg/day (worst case). Total exposure to an animal is the sum of the dermal and oral dose. For example, the worst case dose to a small animal is estimated to be 3.4 mg/kg/day (0.4 + 3.0). Since these dose levels are far below the acute oral LD_{50} for trichlorfon established for laboratory animals (Appendix F, Table 7) mortalities to pets, wildlife, or farm animals would not be expected from the use of trichlorfon to control gypsy moth. The estimated worst case dose levels exceed NOEL values for cholinesterase inhibition for a number of animals (Table 4, Appendix F). Temporary reductions in cholinesterase levels of domestic or wild animals are therefore possible from the use of trichlorfon to control gypsy moths. However, the probability of the worst case dose occurring is low (0.0018), about one chance in 500. The estimated worst case doses to animals are below the teratogenic thresholds by factors greater than 100.

Toxicology data reviewed in Appendix F (p. F-13) indicate that trichlorfon is a possible human mutagen. It also causes cholinesterase depressions at low levels of exposure and causes possible teratogenic effects at very high levels (Table 4, Appendix F).

Estimated realistic and worst case dose levels for the general public and workers that could result from trichlorfon used in gypsy moth eradication and suppression programs are listed in Table 11 (Appendix F). Workers in the mixer/loader group receive the highest possible doses, ranging from 0.046 mg/kg/day (realistic) to 0.20 mg/kg/day (worst case). The highest general public group was made up of individuals who were outside and exposed to a direct application (observer) and eat and drink contaminated food and water. Their estimated exposure levels ranged from 0.012 mg/kg/day (realistic) to 0.174 mg/kg/day (worst case). When the estimated exposure levels to the general public are compared to the ADI for trichlorfon set by the World Health Organization, all realistic dose estimates are below the ADI. EPA is in the process of reestablishing their ADI; therefore WHO's is used which is lower (EPA 1984b). The realistic doses are thus considered to be below threshold dose effects (e.g. birth defects) by margins of safety that are greater than 100. The worst case dose estimates that include consuming food or water containing residues of trichlorfon, as well as all worker exposures, are all greater than the ADI. This indicates that some adverse health effects would occur at these dose levels. Examination of the NOEL values, Tables 4 and 7 (Appendix F), indicates that cholinesterase inhibition could occur at these worst case exposures. However, even these worst case doses are below the teratogenicity threshold by margins of safety greater than 100.

Dermal exposure and possible water contamination resulting from various accident possibilities are discussed in Appendix F (p. F-52 to F-62) and listed in Table 15 (Appendix F). Acute, short term dermal exposures resulting from aircraft spills could range up to 121 mg/kg/day (realistic) to 220 mg/kg/day (worst case). These levels are below dermal LD_{50} values. Dose levels resulting from consumption of water contaminated by an aircraft spill exceed the ADI. A comparison to NOEL values indicates that some short term cholinesterase inhibition could take place if this water were drunk.

Truck accidents could result in very high short term dermal exposures, 10130 mg/kg/day (Table 15, Appendix F), to individuals involved with truck spills. Since this level of exposure exceeds the dermal LD_{50}, such exposure poses a definite hazard. Doses associated with drinking water contaminated by a truck spill should also be considered hazardous because they exceed the ADI. Drinking this contaminated water would result in short term reduced cholinesterase activity. Although the truck and aircraft spills pose a definite hazard to human health, the probability of their occurrence is low: 5.7×10^{-4} for aircraft spills (or less than 1 chance in 1000 aircraft flights) and 1.08×10^{-5} for trucks (or about 1 chance in 100,000).

The risk of cancer resulting from exposure to trichlorfon was evaluated because of the possible mutagenic and carcinogenic potential of trichlorfon (Appendix F, p. F-76 to F-79 and F-94 to F-96). The lifetime risk to an individual receiving the highest worst case dose is 1.34×10^{-7}. This cancer risk is about 10 times lower than the risk of cancer from smoking 2 cigarettes, drinking 40 diet sodas or eating 90 lbs. of charcoal broiled steaks in a lifetime.

Worst case estimates of heritable human mutations resulting from exposure to trichlorfon indicate an individual risk ranging from 1×10^{-7} to 1×10^{-6} (or less than one chance in a million). However, because of the high uncertainty about quantifying the risk of heritable human mutations (currently beyond the state-of-the-art), mitigating measures may want to be considered to minimize exposure to trichlorfon. These would include: not using trichlorfon, limiting trichlorfon use to uninhabited areas, or limiting trichlorfon to sparsely populated areas. Only the first two options reduce risks to an individual. If trichlorfon is used in sparsely populated areas, individual risk would still be in the range of one-in-a-million.

BIOLOGICAL INSECTICIDES

In reviewing the available literature, USDA found 2 reports of any adverse effects attributable to the biological insecticides (Samples and Buettner 1983 and Warren et al. 1984). These reports discuss isolation of vegetative cells and spores of B. t. from a severe skin infection and an ocular (eye) ulcer. The skin infection occurred when a laboratory technician accidentally stuck a needle contaminated with B. t. var. israelensis spores and crystals into his finger. The eye ulcer resulted from accidentally splashing B. t. var. kurstaki into the eye of a farm worker. Although the skin infection involved a B. t. variety that is not used to control gypsy moth, these incidences represent the first reported occurrence of an infection in humans that has been caused by any B. t. variety. These reports need to be viewed with caution because they do not present cause and effect results from a controlled scientific experiment.

The scientific data base is replete with studies describing the safety of these materials to nontarget organisms. No issue or concerns were raised during scoping activities suggesting human health uncertainty with the use of biological insecticides. Furthermore, no human health uncertainty was identified in any review comment letters to the draft EIS. In preparation of this FEIS, USDA identified no relevant data gaps or scientific uncertainty relative to the biological insecticides that would impede a reasonable choice among the alternatives.

Two biological insecticides currently are registered for use against gypsy moth by EPA. These are the bacterium Bacillus thuringiensis Berliner (B. t.) and the gypsy moth nucleopolyhedrosis virus (NPV). B. t. is an aerobic, spore-forming, crystal-producing member of the bacterial genus Bacillus. NPV is a naturally occurring virus of the gypsy moth that causes polyhedrosis or wilting. Field research has been conducted and is continuing on the purification, formulation, and use of NPV. Subsequently, only various formulations of B. t. are currently available for gypsy moth management.

Studies on the fate of B. t. in the environment indicate that B. t. spores will persist in soil for several weeks depending on the soil type, soil flora, and on factors such as pH, moisture, and solar radiation. A study of soils treated with B. t. applied for vegetable pest control concluded that spores can remain viable for long periods (over 3 months), and that the organism can germinate and compete vegetatively in the soil and sporulate successfully under favorable soil conditions (Saleh 1969). The crystal is proteinaceous; degradation by the enzymatic action of soil flora can be presumed.

Although survival of early formulations of B. t. on foliage was limited, present formulations retain residual effectiveness for four to ten days.

In as much as B. t. is exempted from tolerance, no residue analysis on food or feed has been performed when B. t. has been used for forest-insect control (Heimpel 1971).

Laboratory-produced gypsy moth NPV has no degrading effect on the environment in which it is applied. It has a shorter residual persistence on bark and in the soil than the NPV occurring naturally in gypsy moth populations (Lewis et al. 1979).

Toxicology

Biological insecticides must be ingested by the gypsy moth larvae to be effective; therefore, larval mortality is not immediate. Larvae generally cease feeding after ingestion of B. t.; however, mortality may not occur until several days to more than a week later. Recent field projects have demonstrated that a single application of B. t. at a dosage rate of 12 BIU/acre can be effective in achieving the objectives of most suppression projects. More than one application of B. t. may be needed in certain situations to achieve suppression or eradication objectives. Generally speaking, proper B. t. application can be expected to reduce gypsy moth populations by 80 percent and achieve 70 percent foliage protection. A word of caution on the potency of the various formulations of B. t. is warranted: due to the processes used in producing this material, there is a ±30 percent variation in potency. Operational use of B. t. for use in eradication projects is discussed under the IPM alternative.

The gypsy moth NPV must be ingested to be effective. Field studies continue to evaluate the effectiveness of the NPV; however, the material needs further evaluation before being used in operational projects. On the basis of field tests, proper application of gypsy moth NPV has been shown to reduce the residual number of egg masses by 75 percent, and also may reduce egg viability in the succeeding year. NPV also can be expected to achieve 50 to 70 percent foliage protection (Lewis et al. 1979).

In the formulations used for gypsy moth suppression and eradication, B. t. is a lepidoptera-specific insecticide; therefore, only insects in the Order Lepidoptera are affected by it. While lepidopterous larvae other than the gypsy moth may be affected, there will be no effect on beneficial insects such as bees (Lewis et al. 1979).

Test results reported by International Minerals and Chemical Corporation indicate that B. t. has no adverse effect on wildlife (IMC 1969). Doane and Hitchcock (1964) stated that B. t. appeared to cause negligible damage to vertebrate wildlife.

An oral acute toxicity study was conducted with *B. t.* on young adult bobwhite quail. The acute oral medial lethal dosage exceeded 10 gm/kg body weight (IBT 1970b). Five male and 5 female quail were fed 10 g/kg by gavage. A similar group was fed distilled water as control. At the end of the 21-day test period, all animals were sacrificed and subjected to a gross pathological examination. No pathology attributable to the test material was found. Growth rate was similar in the test and control groups.

B. t. administered by mouth as the spore-crystal complex to rats daily for 3 months at rates of 25, 100, and 400 mg/kg/day produced no main function disorders or organ damage. Similar results were obtained in dogs fed 6, 25, and 100 mg/kg/day for 3 months (Fisher and Rosner 1959; Corlett 1961).

Fed to groups of 10 mice (16 to 25 gms.) at the rate of 10 g/kg *B. t.*,(Dipel) caused no mortality. The LD_{50} was beyond 10 g/kg (IBT 1970a). *B. t.* (Dipel) was fed to 3 female mongrel dogs at a dosage of 400 mg/kg/day. The animals were free of any symptoms during the 48-hour observation period (IBT 1970a).

In a test by Briggs and Goodrich (1959), 17 pheasants and 2 partridges, all about 6 weeks old, were divided into 2 groups. One group was fed 1.0 g of *B. t.* per bird per day in 2 gelatin capsules. The control groups were fed 2 empty gelatin capsules daily. No deaths or symptoms of respiratory, alimentary or other disturbances were noted in the group that was fed *B. t.* Two pheasants in the control group died of trauma (due to handling). Birds in both groups exhibited feather color and pattern, bearing, and weight gain that are expected in similar groups of birds in nature. It was concluded that there were no differences in behavior or development between the test and control birds. A long-term study with 6 New Hampshire Red laying hens was conducted over a 23-month period. The hens received a daily dose of *B. t.* ranging from 0.5 to 10 g per bird. Results showed no allergic response, other illnesses, or variations in the expected egg production of the hens. There were no significant differences between the test birds and the birds used as controls. In a 9-week oral toxicity test administered to 24 groups of 10 chicks each, no significant differences were noted between the test and control groups of chicks (Fisher and Rosner 1959).

Eighteen humans each ingested 1 gram of Thuricide daily for 5 days. Complete physical and laboratory examinations were given before the experiment, at the end of the 5-day ingestion period, and 4 to 5 weeks later. Physical examinations included detailed history and records of height, weight, temperature, blood pressure, respiratory rate, and pulse rate immediately after exercise and 30 and

60 seconds thereafter. Evaluations were made of genitourinary, gastrointestinal, cardiorespiratory, and nervous systems. Lab tests included routine urinalysis with qualitative and quantitative urobilinogen determinations (when indicated), complete blood count, sedimentation rate, blood urea nitrogen, glucose, bilirubin and thymel turbidity tests. All subjects remained well during the course of the experiment. All laboratory findings were negative (Fisher and Rosner 1959).

Dermal effects of B. t. were tested by application to shaved flanks and bellies of albino rabbits. Dosages ranged from 20 percent suspensions to 50 mg/animal. After application, half of the treated skin was abraded while the other half was left intact. Readings were made at 24, 48, and 72 hours in one test and up to 3 weeks in another. Other than local, mild erythema (abnormal redness of the skin), no ill effects were noted in any test animal (Fisher and Rosner 1959; Corlett 1961). In another study, dermal application to albino rabbits was made to test allergenicity response. Ten sensitizing doses were applied every other day for 3 weeks. Readings were made 24 hours after each application of B. t. Two weeks after the 10th application, a challenge dose was applied. Only slight erythema and edema were noted. No allergenic response was elicited (Fisher and Rosner 1959). Allergenicity also was tested with guinea pigs following the procedure of Draize. No allergenic response was noted (Fisher and Rosner 1959).

Several acute toxicity tests were conducted on fish. A 4-day toxicity study was conducted with B. t. on rainbow trout and bluegills. Two groups of 10 fish each were placed in water containing B. t. at concentrations of 560 and 1,000 ppm. None of the trout or bluegills died (Fisher and Rosner 1959). Rainbow trout that were 4 inches long were exposed to B. t. at concentrations of 100 to 1,000 ppm for 14 days. No deaths resulted, nor were there symptoms of alimentary or behavioral disturbances evident (Fisher and Rosner 1959). In a test with juvenile coho salmon (1.6 inches long), B. t. was about 1/30 as toxic as DDT. The tests ran for 168 hours with concentrations of 8 to 406 mg B. t. per liter of water. The 48-hour median tolerance limit of the B. t. was about 50 mg/liter (Fisher and Rosner 1959).

Inhalation studies of B. t. were conducted on mice, rats, guinea pigs, and human volunteers. In one test with mice, the animals were exposed to 10 g of B. t. powder for 15 minutes. Dosages were applied 4 times over a period of 6 days. No ill effects were noted and gross pathology was negative (Fisher and Rosner 1959). In tests with rats and guinea pigs, exposure to a 10-percent B. t. preparation for 10 minutes produced no fatalities for the 1-week observation

time. Dyspnea (discomfort) was noted, but recovery was rapid. The animals showed normal weight gain (Fisher and Rosner 1959). Five human volunteers inhaled 100 mg of B. t. powder daily for 5 days. Complete physical examinations before the test, immediately after the test, and 4 to 5 weeks later showed no abnromal conditions in the test subjects (Fisher and Rosner 1959).

Ocular irritation with B. t. was tested in albino rabbits. A dosage of 0.1 cc of a 20-percent suspension was instilled in each eye. One eye was rinsed immediately with isotonic saline. Six animals were tested. The eyes were examined immediately, after 3 hours and 24 hours, and every 24 hours until they appeared normal. Slight redness of the eyelids was noted at 3 and 24 hours. Eye irritation disappeared in 48 hours (Fisher and Rosner 1959).

NPV is an extremely specific virus, affecting only members of the insect Family Lymantriidae. It has been shown to have no effects on other vertebrate or invertebrate organisms.

It is highly unlikely that the registered use of B. t. or NPV as applied during gypsy moth suppression or eradication projects would pose a human health hazard. However, because of the newly reported infections from B. t. occurring in occupationally exposed workers, caution should be exercised when working with the concentrated mixtures.

INTEGRATED PEST MANAGEMENT

An IPM strategy to gypsy moth management includes the integrated use of insecticides, parasite and predator management, the gypsy moth pheromone, release of sterile or partially sterile gypsy moth life stages, and forest stand manipulation. This approach provides a wider range of options in dealing with the gypsy moth problem by providing both short-term and long-term solutions; however, some of this technology still is in the developmental state. Currently, only the biological and chemical insecticides are considered viable components for meeting the objectives of gypsy moth suppression projects. The use of forest stand manipulation, release of sterile or partially sterile gypsy moth life stages, and parasite or predator management need further field evaluation.

Eradication tools in addition to chemical pesticides are being developed. The unique nature of isolated infestations requires that eradication techniques be evaluated in this type of situation. This adds a degree of uncertainty in meeting eradication objectives since little efficacy data exists for some of the IPM components. Certain components

have demonstrated population reduction potential, but several seasons may be necessary to achieve gypsy moth eradication objectives. B. t. was tested operationally in eradication projects for the first time in 1983 at 5 locations. This recent work was done with higher rates of B. t. (16 BIU per acre per application) than have been used in the past, and with as many as 3 aerial applications. Results, although preliminary, are encouraging. Additional experience has been gained with the use of B. t. in conjunction with mass trapping techniques in a similar number of locations. In 1983, approximately 25 percent of the 13,483 acres treated for eradication purposes was treated with B. t. or B. t. and mass trapping. In 1984, 50 percent of 40,000 acres treated for eradication utilized these treatments. Because of positive results on a total of 12 sites where B. t. alone was used and 15 where mass-trapping followed B. t. applications (an aggregate of approximately 24,000 acres), we are gaining added confidence in the efficacy of this biological insecticide for eradication.

The biological effects of an IPM approach will depend on the extent to which the various components are used. An IPM approach encourages the selection of insecticides or other components on the basis of actual needs and management objectives. The biological effects of the registered insecticides as used in an IPM approach have been discussed in the chemical and biological insecticide alternatives section.

Parasites and predators play an integral role in the overall gypsy moth management strategy in generally infested areas. Since it is neither economically or environmentally feasible nor desirable to treat the entire infested area with insecticides, parasites and predators are relied on to reduce gypsy moth populations in areas that are not treated. In the treatment area selection process, areas that support parasite or predator populations sufficient to maintain gypsy moth populations below damaging levels are not identified for insecticide treatment. Treatment is considered only in those high-use and high-value areas where the threat of excessive larval nuisance and host defoliation is immediate, and where parasites, predators, and disease agents are not exerting effective biological pressure on gypsy moth populations.

Manipulation of parasite or predator populations to levels that would exert significant pressure on gypsy moth populations to reduce larval nuisance and host defoliation or mortality entails the timely release of large numbers of laboratory-reared specimens. Since the gypsy moth was introduced into the United States, extensive efforts have been directed to the introduction of parasites and predators. To date, approximately 50 species have been

imported from Europe and Asia with limited degrees of success. The primary problem in the manipulation of parasites and predators is to establish and maintain populations at levels that will contribute to effective biological control.

Grimble (1976) studied the effects of the release of an established larval parasite, Apanteles melanoscelus (Ratz.) (Braconidae), and a pupal parasite, Brachymeria intermedia (Nees)(Chalcidae), on gypsy moth populations in New York. He concluded that the release of A. melanoscelus failed to increase the levels of parasitism by that species. The inundative release of B. intermedia did cause a significant increase in parasitism but only within a 30-chain (0.375 mile) distance of the release points.

In 1982, two new parasites from India were introduced in Delaware. They are A. flavicoxis and A. indiensis, parasites of the Indian gypsy moth. There is no indication that either of these parasites is established.

Between 1973 and 1979, 15 species of exotic parasites and predators from France, India, Spain, Yugoslavia, Japan, and Morocco were released in Pennsylvania to supplement existing populations of established parasites. Total project costs approached $1.5 million. By 1979, there was no evidence of any of these species becoming established. 3/

Since 1970, woodland study sites in New Jersey have been maintained to develop an understanding of gypsy moth population dynamics. During 1978, 402,047 parasites representing nine species were released in these sites. By 1979, a complex system of parasites appeared to be exerting biological pressures against the gypsy moth, one of the more significant parasites being Parasetigena silvestris (Robineau-Desvoidy). 4/

The gypsy moth sex pheromone, disparlure, has shown success in gypsy moth attraction and mating disruption strategies. The USDA and cooperating State agencies have successfully used disparlure- baited traps to delimit gypsy moth population boundaries and to identify isolated infestations. The attractive properties of disparlure make it an invaluable survey tool for locating predamaging gypsy moth populations.

3/ Robert A. Fusco, Pennsylvania Department of Environmental Resources, paper presented at Gypsy Moth Review, Columbus, Ohio, 1979.

4/ Letter from W.W. Metterhouse, N.J. Dep. Agric. to R.G. Doerner, NA, S&PF, USDA FS, dated July 30, 1979.

Disparlure is registered by the EPA. It is recommended for use only in low-level populations to reduce the incidence of gypsy moth mating. The reduction of mating will subsequently reduce the number of egg masses laid, which will help to maintain gypsy moth populations below damaging levels. The registered product, Hercon Luretape , is a disparlure-impregnated tape requiring manual application of forty 2-inch-square tapes per acre in a grid pattern. The registered application rate is 10 to 40 g active ingredient disparlure per acre. A second registered product by Hercon is a disparlure impregnated flake designed for aerial application at rates of 10 to 40 g/acre 5/.

The effectiveness of disparlure as a mating disruptant is density dependent. This means that the lower the level of infestation, the more effective the pheromone, the heavier the infestation, the less effective the pheromone. Therefore, the level of infestation must be determined before treatment to ensure the greatest mating disruption.

The use of disparlure to meet the objectives of foliage protection, larval nuisance reduction, and total population suppression requires further investigation. In low-level, isolated populations like those treated in APHIS/State eradication projects, disparlure baited, high-capacity traps, when set in dense arrays, show some potential for gypsy moth control; however, populations must be at extremely low levels. In heavy populations as proposed for treatment in suppression projects, disparlure alone is not effective.

Disparlure may prove feasible to further reduce populations that have been suppressed with insecticides or another component of an IPM program to meet suppression or eradication project objectives. The effects of disparlure also may occur in the 2nd year after application if the material successfully reduced population levels. These techniques need to be further developed.

The use of forest stand manipulation to suppress gypsy moth populations in high-value forest stands has been suggested in the past. However, this method has not proven biologically effective. In suburban woodlands, stand

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5/ Letter from A. R. Quisumbing, Health-Chem Corporation to Noel F. Schneeberger, NA, S&PF, USDA, FS, dated November 2, 1983.

manipulation is considered feasible to reduce gypsy moth impacts; less preferred hosts could be encouraged or even planted. Tree species that are less susceptible to gypsy moth defoliation include black walnut, white ash, catalpa, flowering dogwood, American holly, tulip-poplar, locust, sycamore, juniper, and balsam fir. In 1983, the USDA Forest Service initiated a research effort to study the use of silvicultural methods to control gypsy moth.

The release of large numbers of sterile or partially sterile male moths to reduce gypsy moth populations is a potential component of IPM. Research and development is continuing on the effectiveness of using sterile or partially sterile male moths to control gypsy moth populations. At the APHIS Otis Methods Development Laboratory, evaluations continue on refining male moth mass-rearing techniques and evaluating competitiveness of irradiated male moths.

A field test in which sterile male moths were used was performed in Michiganin 1980 and 1981. Monitoring of this site in 1982 and 1983 indicates that this infestation is now eradicated. APHIS will monitor this site for 1 more season before assessing final results. Development of the sterile and partially sterile male moth technique is targeted for use in isolated infestations outside of the Northeast where the technique might be useful in an eradication strategy. There is no threat of human sterility should persons come into contact with sterile or partially sterile gypsy moth adults. Implementation of chemical and biological insecticides in an IPM approach may result in physical effects similar to those described under the chemical insecticide and biological insecticide alternatives. Implementation of parasite and predator management, the gypsy moth pheromone, and release of sterile or partially sterile male moths will not cause any adverse impact on the soil, water, or air in the treatment areas.

Forest stand manipulation through harvesting and thinning methods could entail favoring less susceptible trees by removing preferred hosts or even by planting less favored hosts. Such activity might result in some soil erosion and silting of adjacent streams. Soil disturbances are temporary and often last no more than 2 or 3 months depending on the time of the year.

An IPM approach favors the increased use of alternative means of suppression over chemical insecticides and the use of those methods of suppression that create minimal impact on the environment while meeting project objectives.

PUBLIC NOTIFICATION AND INVOLVEMENT

In accordance with the NEPA process, the USDA encourages public involvement in the development of gypsy moth suppression and eradication projects. Public notification procedures relevant to these projects include:

- Providing public notice of scoping activities.

- Making EIS and related documents available to inform those agencies, groups, and individuals who may be interested in or affected by proposed actions. Copies of this Final EIS can be obtained by contacting Thomas N. Schenarts, USDA Forest Service or Robert L. Williamson, USDA APHIS. Addresses and phone numbers are listed on the first page of this document. Copies of past gypsy moth EIS's may also be obtained until available supplies are depleted. Thereafter, a fee will be assessed for making duplicate copies.

- Announcement of treatment dates to make it possible for anyone who has questions or concerns about adverse insecticide sensitivity to seek medical advice and adequate shelter that will avoid exposure during or after treatment or to leave the area to be treated until all danger of exposure has passed.

In addition, State and Federal agencies that cooperate with USDA and the Forest Service will actively seek public participation and involvement at the local level. The purpose of this public involvement process is to:

(1) Explain the proposed action and its need.

(2) Discuss the consequences (if any) of the proposed action.

(3) Solicit identification of local issues and concerns or individuals particularly sensitive to the insecticides planned for use so that appropriate mitigating measures can be developed.

(4) Stimulate discussion of alternative measures and their consequences.

(5) Guide the environmental analysis process.
For gypsy moth suppression activities on private land, some States may allow land owners the option of not participating in proposed suppression projects. This option is based on State law or policy. Because of the

objective of eradication projects, residents do not have the option of having their property deleted from the proposed treatments. As previously discussed, mitigating measures will be employed to minimize the concerns of those residents who are unable to opt out of eradication projects.

Specific public participation and notification procedures relative to individual gypsy moth suppression and eradication projects will be developed during site-specific environmental analyses, and conducted in accordance with NEPA.

LIST OF PREPARERS

Name: Noel F. Schneeberger

Position: Entomologist, USDA Forest Service, State and Private Forestry, Forest Pest Management, Morgantown, WV 26505.

Experience and Education: Seven years service with USDA Forest Service. In current position since 1982. Previous assignment with USDA Forest Service was with the Application Group, Expanded Gypsy Moth Research, Development and Application Program, Hamden, CT. From 1976-1978 was Forest Pest Specialist with South Dakota Division of Forestry. Holds Bachelor degree from Wittenberg University, Springfield, OH, and Masters degree in Forest Entomology from Duke University, Durham, NC.

EIS Responsibility: Coordinated the environmental analysis for USDA Forest Service suppression projects. Coordinated development and compilation of the FEIS and Risk Analysis (Appendix F).

Name: Robert D. Wolfe

Position: Staff Pathologist, USDA Forest Service, State and Private Forestry, Forest Pest Management, Broomall, PA 19008.

Experience and Education: In present position since 1976. Nineteen years employment with the USDA Forest Service. Previous assignments were at Macon, GA, Alexandria, LA, and Asheville, NC. Holds a B.S. degree in Biology and a M.F. in Forest Pathology from Duke University, Durham, NC.

EIS Responsibility: Coordination of FEIS review, printing, distribution and filing with EPA. Major participation in environmental analysis for Forest Service suppression projects, and compilation of the FEIS. Coordinated Forest Service response to DEIS review comment letters. Participated in development of the Risk Analysis (Appendix F).

Name: Gary E. Moorehead

Position: Staff Officer, USDA APHIS, Plant Protection and Quarantine, Hyattsville, MD 20782.

Experience and Education: In current position since 1980. Twenty-five years service with APHIS, Plant Protection and Quarantine Program, sixteen of which involve Methods Development activities on biological control, IPM and gypsy moth. Holds a B.S. degree in Botany from Iowa State University and has done graduate work in Entomology at Purdue University.

EIS Responsibility: Coordinated environmental analysis for USDA APHIS eradication projects and assisted in the compilation of the FEIS. Responsible for compiling APHIS response to DEIS review comment letters. Participated in development of the Risk Analysis (Appendix F).

Name: John Neisess

Position: Pesticide Specialist, USDA Forest Service, State and Private Forestry, Forest Pest Management, Washington, D.C.

Experience and Education: In current position since 1979. Responsible for pesticide impact assessment. Previously, research chemist with the USDA Forest Service, Pacific Northwest Forest Experiment Station, responsible for pesticide development, particularly microbials. Holds a Ph.D. in chemistry from Oregon State University.

EIS Responsibility: Major participant in development and compilation of the Risk Analysis (Appendix F). Reviewed toxicological information, developed exposure scenarios, and cancer risk analysis.

Name: John H. Ghent

Position: Entomologist, USDA Forest Service, Region 8, Forest Pest Management, Asheville, NC 28804.

Experience and Education: In current position since 1976. Eight years employment with USDA Forest Service. Holds a B.S. degree in Biology from Catawba College, Salisbury, NC and M.F. degree in Forest Protection from Duke University, Durham, NC.

EIS Responsibility: Participant in rewriting and updating of the Risk Analysis (Appendix F).

Name: Charles I. Shade

Position: Public Affairs Specialist, USDA Forest Service, State and Private Forstry, Forest Pest Management, Washington, D.C.

Experience and Education: From 1980 to 1984, Public Affairs Officer, USDA Forest Service, Oxford, Mississippi; 1970 to 1980, Public Information Director, Mississippi Employment Security Commission, Jackson; 1955 to 1970, Operations Manager, WDAM-TV, Hattiesburg, Mississippi. A.B. magna cum laude, A.M., Harvard University, Ph.D., Nara University, Nara, Japan.

EIS Responsibility: Editorial and Design for the Risk Analysis (Appendix F).

The preparers of the FEIS and Risk Analysis (Appendix F) were concerned whether toxicological information and cancer risk analysis procedures had been properly interpreted and used in the Risk Analysis. Because of this concern, the Forest Service initiated a contract with Labat-Anderson Incorporated (1111 19th Street North, Suite 600, Arlington, Virginia 22209) for a technical review of a working draft and the Draft Supplement to the FEIS. The following individuals provided review of the subject documents under the contract with Labat-Anderson, Inc.:

Name of Reviewer	Special Interest
Dr. David Brusick Director, Department of Molecular Toxicology Litton Bionetics Kensington, MD 20895	Mutagenicity
Dr. Richard Wilson Chairman, Department of Physics Harvard University Cambridge, MA 02138	Cancer risk analysis
Dr. Frank Dost Department of Agricultural Chemistry Oregon State University Corvallis, OR 97331	General toxicology and risk analysis
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GLOSSARY

Acceptable Daily Intake (ADI)
: The maximum dose of a substance that is anticipated to be without lifetime risk to humans when taken daily.

Acephate
: Organophosphate insecticide; the active ingredient found in insecticide formulations sold under the trade name Orthene®.

Acetylcholine
: A compound that is released at many autonomic nerve endings. It is believed to function in the transmission of the nerve impulse.

Acetylcholinesterase
: An enzyme released at nerve endings in order to accelerate hydrolysis of acetylcholine thereby ending nerve stimulation after an impulse has passed.

Active ingredient (AI)
: The effective part of a pesticide formulation, or the actual amount of the technical material present in the formulation.

Acute toxicity
: The toxicity of a compound when given in a single dose or in multiple doses over a period of 24 hours or less.

AI
: Abbreviation for active ingredient.

APHIS
: Animal and Plant Health Inspection Service. The USDA agency responsible for regulating materials which have potential for artificially moving gypsy moth out of quarantined areas and for eradicating isolated infestations of gypsy moth.

Apiary
: A place where bees are kept. Bee hives.

Arthropods
: Major group of invertebrate animals belonging to the phylum Arthropoda. This group includes insects, spiders and crustaceans.

Artificial spread
: Term used to described the spread of gypsy by other than natural means; e.g. hitch-hiking insect stages on recreational vehicles, campers, cars, nursery stock, household goods, etc..

Bacillus thuringiensis
: Scientific name of a bacterium that is pathogenic to the larval stage of many lepidopterous insects. The active ingredient in biological insecticides sold under such names as Dipel®, Bactospeine® and Thuricide®.

B. t.
Abbreviation for *Bacillus thuringiensis*.

Buffer zones or areas
Usually set around sensitive areas such as lakes, streams or ponds that are not directly treated with insecticides; or areas set around the same, including people who object to chemical insecticides, that are treated instead with microbial insecticides such as *B. t.* or gypsy moth NPV. In some cases, may refer to areas actually treated, such as treatment of buffer zones along roads.

Caddisfly
A small moth-like insect. The larvae live in fresh water in portable cases they construct around themselves. Member of order Trichoptera.

Carbaryl
Carbamate insecticide; the active ingredient in insecticide formulations sold under the tradename Sevin .

Carcinogenicity
Tendency of a substance to cause cancer.

Chitin
A semi-transparent horny substance forming the principal component of crustacean shells, insect exoskeletons and the cell walls of certain fungi.

Chitinase
An enzyme that hydrolyzes chitin.

Cholinesterase
See acetylcholinesterase.

Chronic toxicity
The effect of a compound on test animals when exposed to sublethal amounts continually. Usually daily exposures over a period of time: weeks, months or years.

Collembola
Springtails. Primitive, wingless group of insects commonly found in soil and duff.

Copepods
Usually, minute freshwater and marine crustaceans belonging to the order Copepoda.

Corixids
Group of aquatic insects, usually freshwater, that feed on algae and other minute aquatic organisms; belong to the family Corixidae.

Crustaceans
: Large group of mostly aquatic arthropods belonging to the class crustacea and characterized by a chitinous or calcareous and chitinous exoskeleton. Members of this group include Copepods, water fleas, shrimps and wood lice, among others.

Cyclops
: Scientific name (genus) of a group of Copepods.

DEIS
: Draft Environmental Impact Statement

Diflubenzuron
: The active ingredient of insecticide formulations sold under the trade name Dimilin . Acts as a growth regulator by interfering with chitin synthesis and prevents gypsy moth from successfully completing their molting phases.

Dimilin W-25®
: Commercial wettable powder formulation of diflubenzuron registered for use against gypsy moth.

Dipel®
: Trade name of biological insecticide formulations containing the bacterium Bacillus thuringiensis.

Disparlure
: Commercially synthesized female gypsy moth sex pheromone. Disparlure is used to disrupt mating by making it difficult for male moths to locate female moths.

Dosage rate
: Quantity of a toxicant applied per unit area. Usually expressed as oz. or lbs. active ingredient per acre.

Dylox®
: Trade name of chemical insecticide formulations containing the active ingredient trichlorfon.

EC_{50}
: Median effective concentration; it is the concentration (ppm or ppb) of the toxicant in the environment (usually water) which produces a designated effect to 50 percent of the test organisms exposed.

EIS
: Environmental Impact Statement.

Environmental analysis
: Procedure defined by the National Environmental Policy Act of 1969 whereby the environmental impacts of a planned action (in this case gypsy moth suppression and eradication projects) are objectively reviewed.

EPA
U.S. Environmental Protection Agency.

Eradication projects
Projects whose objective is to eliminate gypsy moth infestations which were started as a result of artificial movement of gypsy moth life stages from generally infested areas.

Exclusion areas
Areas where product label prohibits the use of an insecticide, or areas identified during public involvement process as no-treatment areas.

FEIS
Final Environmental Impact Statement

Foliage protection
Tree foliage is considered to be protected if the amount of defoliation that occurs is not severe enough to cause the tree to refoliate or produce a new set of leaves. Generally one of the major objectives in suppression projects.

Formulation
The form in which a pesticide is packaged or prepared for use.

Frass
Insect solid excrement.

FS
Forest Service. The USDA agency responsible for gypsy moth suppression projects.

Generally infested area or areas
That area, from Maine to northern Virginia and eastern West Virginia in which the gypsy moth is considered to be permanently established. Also includes an area in central Michigan in which gypsy moth is permanently established and where APHIS is no longer persuing eradication activities.

Gypchek
USDA laboratory prepared and refined gypsy moth NPV product. Used as a biological insecticide.

Half-life
The time required for half the amount of substance (such as an insecticide) in or introduced into a living system to be eliminated whether by excretion, metablic decomposition, or other natural process.

Hemiptera
True bugs. Group of insects with semi-toughened forewings and sucking mouth parts.

Hymenoptera
: A large order of insects comprised of the ants, bees, sawflies and wasps. The typical adult each have four membranous wings and chewing type mouthparts.

Instar
: The term for a insect before each of the molts (shedding of its skin) it must go through in order to increase in size. Upon hatching from its egg, the insect is in instar I and is so called until it molts, when it begins instar II, etc.

Invertebrate
: Major group of animals of which arthropods are members; characterized by the lack of backbone and spinal column.

IPM
: Integrated Pest Management.

Isolated or remote infestation
: As pertains to gypsy moth, any infestation(s) occurring outside of generally infested area resulting from artificial spreadof insect life stages, as opposed to natural spread of the insect. Once established, isolated infestations may spread or expand naturally if they are not eradicated.

Larva (plural larvae)
: An insect in the earliest stage of development, after it has hatched and before it changes into pupa; a caterpillar, maggot, or grub.

LC_{50}
: The median lethal dose; the size of a single dose of a chemical necessary to kill 50 percent of the organisms in a specific test situation. It is usually expressed in the weight of the chemical per unit of body weight (mg/kg). It may be fed (oral LD_{50}), applied to the skin (dermal LD_{50}), or administered in the form of vapors (inhalation LD_{50}).

LD_{50}
: Median lethal dose, is the milligram of toxicant per kilogram of body weight (mg/kg) lethal to 50 percent of the test animals to which it is administered under the conditions of the experiment.

Lepidoptera
: A large order of insects, including the butterflies and moths; characterized by four scale-covered wings and coiled sucking mouthparts.

mg/kg/day
: Milligrams per kilogram of body weight per day.

mg/kg
: Milligrams per kilogram; used to designate the amount of toxicant required per kilogram of body weight of test organisms to produce a designated effect; usually the amount necessary to kill 50 percent of the test animals. One mg/kg = 1 ppm. One mg = 0.000035 ounce, and 1 kg = 2.2 pounds.

Mutagenicity
: The capacity of a substance to cause changes in genetic material.

Natural spread
: Opposite of artificial spread; spread of gypsy moth through natural means, for example young larvae carried on the wind or older larvae walking to new food sources. Natural spread of gypsy moth occurs from generally infested areas, or from permanently established isolated infestations.

NEPA
: National Environmental Policy Act of 1969, Public Law 91-190.

NOEL
: The No Observable Effect Level. In a series of dose levels tested, it is the highest level at which no effect is observed, i.e., safe in the species tested.

Notonectids
: Group of predaceous aquatic insects belonging to the family Notonectidae. Commonly called backswimmers.

NPV
: Nucleopolyhedrosis virus. In this case, naturally occurring virus specific to gypsy moth, and common in heavy gypsy moth populations. The active ingredient in the biological insecticide Gypchek.

Orthene®
: Commercially produced chemical insecticide formulation containing the active ingredient acephate.

Parasite
: Any animal that lives in, on, or at the expense of another.

Pheromone
: As pertains to gypsy moth, chemical produced and emitted by female moths to attract male moths for mating.

Phytotoxic
: Poisonous or harmful to plants.

Plecoptera
: Stoneflies. Group of insects, the nymphs of which are aquatic and mostly phytophagous.

Ppb

Parts per billion; the number of parts of a substance in question per billion parts of a given material. One ppb = 1 ug/liter (water or air).

Ppm

Parts per million; the number of parts of a substance in question per million parts of a given material. (1 ounce of salt in 62,500 lbs of sugar). One ppm = 1 mg/kg (on a weight basis) = 1 mg/liter (water or air).

Predator

An animal that preys on others.

Pupa (plural pupae)

The immobile, transformation stage in the development of an insect that, as an adult, is completely different in its appearance compared to what it looked like when it hatched from its egg. Examples include beetles, flies, moths, and wasps.

Quarantine area(s)

See regulated area(s).

Refoliation

Term used to describe a new flush of leaves in mid-season. In gypsy moth projects, if a tree has to refoliate, then the objective of foliage protection was not achieved.

Regulated area(s)

Areas where gypsy moth is permanently established and reproducing, and from which APHIS regulates the movement of materials such as household goods, nursery stock, and other commodities in order to prevent artificial movement of gypsy moth life stages to infested areas of the United States.

Regulatory programs

As pertains to gypsy moth, APHIS programs designed to reduce artificial spread from regulated areas and to eradicate isolated infestations of gypsy moth.

Remote infestations

See isolated infestations.

RPAR

Rebuttable Presumption Against Registration. EPA process for reviewing and subsequently approving or withdrawing registration of pesticides.

Scoping Session or activities

As defined under the National Environmental Policy Act - an early and open process for determining the scope of issues to be addressed and for identifying the significant issues related to a proposed action. This may include public meetings whereby significant issues are identified, or may simply be letters of inquiry to interested agencies, groups or individuals.

Sevin 4 Oil®
: Commercial insecticide formulation containing the active ingredient carbaryl.

Sevin 80 S®, Sevin Sprayable®, Sevin XLR®
: See Sevin 4 Oil.

Suppression projects
: Projects administered by USDA Forest Service, in cooperation with State or Federal agencies, designed to relieve high gypsy moth populations in high-value high-use areas or to prevent tree mortality in forested areas. Also includes comparable projects on National Forest System lands.

Tachinidae
: Family of flies, the larvae of which are parasitic.

Teratogenicity
: The capacity of a substance to cause anatomical, physiological, or behavioral defects in animals exposed during embryonic development.

Thuricide®
: Commercial biological insecticide formulation containing the active ingredient Bacillus thuringiensis.

Trichlorfon
: Active ingredient found in chemical insecticide formulations sold under the tradename Dylox®.

USDA
: United States Department of Agriculture.

USDI
: United States Department of the Interior.

APPENDIX A

1983 SCOPING PROCESS: AGENCIES, ORGANIZATIONS, AND INDIVIDUALS CONTACTED*

* The mailing list for the 1983 Scoping Process is contained in the 1984 FEIS and therefore is not reproduced here. Copies of the 1984 FEIS may be obtained by writing to the USDA Forest Service or APHIS address listed on the cover page of this document.

APPENDIX B

FEIS MAILING LIST AS SUPPLEMENTED-1985*

* The mailing list for the 1984 FEIS is contained in that document and has been replaced with an expanded list for distribution of this FEIS as supplemented. Copies of the 1984 FEIS containing the original mailing list may be obtained by writing to the USDA Forest Service or APHIS address listed on the cover page of this document.

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APPENDIX C

REVIEW OF EXPERIMENTAL EVIDENCE ON THE MUTAGENICITY OF N-NITROSOCARBARYL

Retyped verbatim from the Carbaryl Decision Document,
December 1980.
U.S. Environmental Protection Agency

REVIEW OF EXPERIMENTAL EVIDENCE ON THE MUTAGENICITY OF N-NITROSOCARBARYL

Carbaryl has been shown in vitro to react with sodium nitrite under acidic conditions (pH 1) to form N-nitrosocarbaryl (Eisenbrand et al., 1974). Because nitrite is present in human saliva and food products, the formation of nitrosocarbaryl in stomach physiology is possible, in view of the widespread use of carbaryl. Rickard (1979) demonstrated the in vitro formation of nitrosocarbaryl in the stomach of rats and guinea pigs. When guinea pigs were given either simultaneous intubation of carbaryl (1 umol) and sodium nitrite (1160 umol), or when these components were mixed with feed, approximately a 1.5 percent yield of nitrosocarbaryl was detected. The formation of this nitroso derivative was dependent on the amount of nitrite and the pH, and not particularly by the amount of carbaryl present. Increasing the amount of carbaryl from 0.025 to 2.5 umol did not increase the yield of the nitroso compound. In rats, the stomach pH (3.5-5.5) is higher than in guinea pigs (pH 1.5), and in that species a very low yield of nitrosocarbaryl was found (0.02 percent) at the same concentrations of nitrite and carbaryl.

Nitrosocarbaryl has been shown to be strongly mutagenic in bacteria. Blevins et al. (1977) found that the base-pair substitution sensitive Salmonella strains TA 100 and TA 1535 were reverted by this without metabolic activation. The reversion frequency in TA 100 was increased by approximately 1.6-fold at 1.15 ug/plate and 6-fold at 11.5 ug/plate, and TA 1535 by about 3-fold at 76-fold at 11.5 ug/plate. Nitrosocarbaryl was not as active on the frameshift sensitive strains TA 98, TA 1537, and TA 1538. Marshall et al. (1976) found that nitrosocarbaryl increased the number of histidine-independent colonies of TA 1535 by approximately 6-fold at 0.5 ug/plate and by 367-fold at 50 ug/plate without metabolic activation. Marshall et al. also found nitrosocarbaryl to be slightly active (above 6-fold over background values) on the frameshift sensitive strains TA 1537 and TA 1538 at 50 ug/plate. Both Blevins et al. (1977) and Marshall et al. (1976) found that the mutagenic activity of nitrosocarbaryl was dose-related.

Elespuru and coworkers (1974) measured the induction to novobiocin resistance in Haemophilus influenzae. These authors found that nitrosocarbaryl was approximately an order of magnitude more potent than the mutagen N-methyl-N'-nitrosoguanidine (MNNG). In Escherichia coli nitrosocarbaryl was also more potent in the induction to arginine prototrophy than MNNG (Elespuru et al., 1974). Uchiyama et al. (1975) found mutagenic activity as tested by the ability to cause reversion at the tryptophan locus in Escherichia coli (data not quantitated).

Generally, metabolic activation was not required for the mutagenic response of nitrosocarbaryl. For example, when Marshall et al. (1977) incorporated the S-9 fraction in the Salmonella assay, a decrease in mutagenic activity was observed. Greim et al. (1977), however, found an increase in mutagenicity after metabolic activation by mouse-liver microsomes.

Siebert and Eisenbrand (1974) reported that nitrosocarbaryl was active in causing mitotic gene conversion in Saccaharomyces cerevisiae. Incubation for 2 hours on 1 ppm of nitrosocarbaryl increased the relative conversion frequency 3-fold for the ade-2 locus and 5-fold for the trp-5 locus, and at 30 ppm increases were 139-fold for the ade-2 locus and 885-fold for the trp-5 locus. In this study, a dose-related effect was shown using 5 concentrations of nitrosocarbaryl. Regan et al. (1976) demonstrated that nitrosocarbaryl was able to induce DNA damage in culture human cells as measured by unscheduled DNA synthesis. In addition, by using methyl labeled [^{14}C] and ring labeled [^{3}H] nitrosocarbaryl, Regan et al. (1976) found that the ^{14}C label was associated with cellular DNA, whereas the ^{3}H label was not. Because nitrosocarbaryl has been observed to cause reversion of base-pair substitution sensitive strains (TA 100, TA 1535), these results suggest that the nitrosocarbaryl molecule was split and the resultant methyl group could alkylate DNA and cause base-pair substitution type mutations.

Ishidate and Odashima (1977) reported several chromosome aberrations (80 percent aberrant cells) in Chinese hamster cells 24 hours after exposure to nitrosocarbaryl (0.015 mg/ml). The toxicity of nitrosocarbaryl was not reported.

APPENDIX D

EPA CARBARYL DECISION DOCUMENT, DECEMBER 1980: SUMMARY OF CONCLUSIONS

CARBARYL DECISION DOCUMENT, DECEMBER 1980
Office of Pesticides & Toxic Substances
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A. Summary of Conclusions*

1. Teratogenic and Fetotoxic Effects. Based on the weight of evidence of currently available studies which are valid and interpretable, the Agency has concluded that a rebuttable presumption on the basis of carbaryl-related teratogenic and fetotoxic effects is not warranted at this time. In the Agency's judgment, the extremely high doses of carbaryl used to elicit effects in the developing organism, coupled with the positive correlation of maternal and fetal toxicity in the multiple species tested (the dog being a possible exception), do not indicate that the pesticide carbaryl constitutes a potential human teratogenic or reproductive hazard under proper environmental usage. However, the Agency is considering whether another study in dogs should be conducted, with special attention paid to sufficient numbers of animals in the dose groups, the condition of the bitches throughout the period of dosing, and maternal and fetal blood levels of the compound.

2. Mutagenic Effects. Based on the weight of extensive existing evidence, the Agency has determined that the current data base does not support a conclusion that carbaryl poses a mutagenic hazard to humans. Due to the weak mutagenic responses which have measured, and due to the suggestive rather than conclusive nature of the evidence available as to the potential of carbaryl to reach the mammalian germinal tissues, the Agency believes that general exposure-reduction measures typical of those already on many of the labels, are appropriate and will be pursued prior to any further RPAR review. A rebuttable presumption on the basis of carbaryl-related mutagenic effects is therefore not warranted at this time.

3. Oncogenic Effects. Based on the weight of existing evidence, the Agency has concluded that the current data base does not indicate that carbaryl poses on oncogenic hazard to humans. A rebuttable presumption on the basis of carbaryl-related oncogenic effects is therefore not warranted at this time.

4. Neurotoxicity. Based on available evidence, the Agency has concluded that carbaryl does not pose a human health hazard in terms of neurotoxic effects. A rebuttable presumption on the basis of neurotoxicity is therefore not warranted at this time.

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* From: With oral approval from EPA, retyped verbatim in order to produce a clear copy.

5. Viral Enhancement. The Agency's determination at this juncture is that research into viral enhancement as a possible adverse effect of exposure to carbaryl is preliminary in nature and that current information does not constitute a basis on which to conclude that carbaryl poses a human hazard in this area. A rebuttable presumption on the basis of viral enhancement is therefore not warranted at this time.

6. Overview--Determining Considerations. Recognizing that the data base on any chemical is necessarily a continuum, the Agency's determination not to proceed with an RPAR action against carbaryl at this time takes into account a number of considerations in connection with the present toxicological picture of the pesticide. As has been pointed out, the current data base under review is extensive, more extensive than has ordinarily been the case for pesticides which have come under Agency review. This is particularly true for teratogenicity/fetotoxicity and mutagenicity, which are the toxicological areas of primary concern, and it is unlikely that resource-intensive RPAR procedures would surface data not already in the Agency's possession via other channels.

Although the current data base is extensive, risk data are not unequivocal, and study results, again in the areas of teratogenicity/fetotoxicity and mutagenicity, have been inconsistent. The current toxicological picture of carbaryl thus reflects a degree of uncertainty. It is in the face of such uncertainty that the Agency must determine whether or not to proceed with an RPAR action and the detailed risk/benefit analysis the RPAR process is intended to implement. In the case of carbaryl, consideration of the overall weight of current evidence leads the Agency to conclude that the responsible call is not to initiate RPAR proceedings at this juncture but rather to address the concerns at issue via the recommendations made below. Should further review data indicate that current use patterns of the pesticide pose unreasonable adverse effects to human health or the environment, however, the Agency will re-open the case of carbaryl as an RPAR candidate.

B. Recommendations

Because the Agency has concluded that a rebuttable presumption against registration and continued registration of pesticide products containing carbaryl is not warranted at this time, the Agency's recommendation is that carbaryl be returned to the registration process. This recommendation is made with the following stipulations: 1) that a FIFRA sec. 3(c) (2) (B) action be considered for additional data on the effects of carbaryl, possible including another study of the teratogenic and fetotoxic effects of carbaryl in dogs 2) that appropriate label changes be implemented according the forthcoming negotiations between the Agency and registrants to ensure that exposure is minimized.

APPENDIX E

HISTORY OF GYPSY MOTH ERADICATION

Appendix E
History of Gypsy Moth Eradication

Location	Year of Initial Treatment	Approximate Acreage	Treatment (Number of Applications)	Status	Remarks
Calhoun County, MI (Duck Lake)	1967	15,000	carbaryl (2)	Eradicated	Remains uninfested.
Jefferson County, PA	1972	625	carbaryl (2)	Eradicated	Overrun by natural spread.
Isabella County, MI	1973	14,386	carbaryl (2)	Infested	Center of developmental activities in '74 & '75. Eradication abandoned '76.
Lorain County, OH	1973	300	trichlorfon (2)	Eradicated	Remains uninfested.
Smyth County, VA	1974	1,000	carbaryl (2)	Eradicated	Remains uninfested.
Forsyth County, NC	1974	640	carbaryl (2)	Eradicated	Remains uninfested.
Monroe County, NY	1974	440	carbaryl (2)	Eradicated	Overrun by natural spread.
Macomb County, MI	1975	500	carbaryl (2)	Eradicated	Overrun by natural spread.
Cook County, IL (Palos Park)	1976	1,000	carbaryl (2)	Eradicated	Remains uninfested.
Outagamie County, WI (Appleton)	1976	40	trapping	Eradicated	Remains uninfested.
Berrien County, MI (Hager Twp.)	1977	2,750	Dimilin (2)	Eradicated	Area just outside treatment block used in '81 for sterile release project.
Washtenaw County, MI (Lodi & Pittsfield Twps.)	1977	3,500	Dimilin (2)	Eradicated	Overrun by natural spread.
Santa Clara County, CA (San Jose)	1977	2,554	Dimilin (2)	Eradicated	Remains uninfested.
Avery County, NC	1979	724	Dimilin (2)	Eradicated	Remains uninfested.
Clark & Loudon Counties, VA and Jefferson County, WV	1979	8,582	Dimilin (2)	Eradicated	No moths trapped '80 & '81; Overrun by natural spread.
King County, WA (Renton)	1979	400	acephate (2)	Eradicated	Remains uninfested.
Waukesha County, WI	1979	337	Gypcheck (2)	Infested	
" "	"	420	Disparlure		
" "	1981	100	Disparlure	Infested	17 moths in '83.
" "	"	425	mass trapping	Infested	14 larvae in '81.
" "	"	10	carbaryl	Infested	64 larvae in '81.
" "	1982	425	mass trapping	Infested	19 moths in '82.
" "	1983	300	mass trapping	Infested	4 moths in '83.
" "	1984	35	B.t. (2)	Infested	28 moths trapped in '84.
" "	"	190	mass trapping		
Montgomery County, OH (Kettering)	1980	340	trichlorfon (2)	Eradicated	Remains uninfested.
Ottawa County, OH (Catawba Island)	1980	200	trichlorfon (2)	Infested	2 moths trapped in '81.
" " " "	1982	84	carbaryl (2)	Eradicated	Remains uninfested.
Calhoun County, MI (Battle Creek Twp.)	1980	1,000	carbaryl (2)	Eradicated	Remains uninfested.
Van Buren County, MI (Pine Grove Twp.)	1980	460	carbaryl (2)	Eradicated	Remains uninfested.

McHenry County, IL (McHenry)	1980	40	B.t. (2)		
" " "	"	300	mass trapping		
McHenry County, IL (McHenry)	1981	100	B.t. (2)		
" " "	"	300	mass trapping	Eradicated	No moths trapped in '81-82.
Lake County, IL (Kildeer)	1980	30	carbaryl (2)	Eradicated	No moths trapped in '81-82.
Floyd County, VA	1980	3,055	Dimilin (2)	Eradicated	11 moths trapped in '80.
" " "	"	50	B.t. (2)	"	B.t. used around lake of same block.
" " "	1981	1,315	Dimilin (2)	Infested	1 moth trapped in treatment block.
" " "	1982	4,632	Dimilin (2)	Infested	5 moths trapped in treatment block in '82.
Lunenburg County, VA	1981	213	Dimilin	Infested	5 moths trapped in '81.
" " "	"	20	carbaryl		
" " "	1982	300	Dimilin (2)	Eradicated	No moths trapped in '82.
Lake County, IL (Diamond Lake)	1981	3	carbaryl (2)	Eradicated	No moths trapped in '81-82.
Lake County, IL (Lincolnshire)	1981	35	carbaryl (2)	Eradicated	Remains uninfested.
DuPage County, IL (Wheaton)	1981	50	carbaryl (2)	Infested	Area had not been delimited.
" " " "	1982	300	B.t. (2)	Infested	103 moths trapped in treatment block in '82.
" " " "	"	300	mass trapping		
" " " "	1983	230	B.t. (2)	Infested	5 moths trapped in '83.
" " " "	"	800	mass trapping		
" " " "	1984	80	mass trapping	Eradicated	No moths trapped in '84.
Oakland County, MI (Bloomfield Twp.)	1981	552	carbaryl (2)	Eradicated	Overrun by natural spread.
Wayne County, MI (Livonia Twp.)	1981	316	carbaryl (2)	Eradicated	Overrun by natural spread.
Kalamazoo County, MI (Cooper Twp.)	1981	643	carbaryl (2)	Eradicated	Remains uninfested.
Kent County, MI (Caledonia Twp.)	1981	530	B.t. (2)		
" " " "	1981	500	disparlure	Infested	
" " " "	1982	517	carbaryl (2)	Eradicated	Remains uninfested.
Berrien County, MI	1981	400	sterile males		No native moths trapped in '82 & '83.
" " " "	1982	400	sterile males	Eradicated	
Stark County, OH (Lake Twp.)	1981	355	carbaryl (2)	Eradicated	Remains uninfested.
Marion County, OR (Salem)	1981	20	acephate	Eradicated	Remains uninfested.
Orange County, CA	1981	10	carbaryl (3)	Eradicated	Remains uninfested.
Lancaster County, NE	1981	1,000	traps/carbaryl		
" " " "	1982	600	mass trapping	Eradicated	Remains uninfested.
King County, WA (Lincoln Pk., Ravenna Pk. and Mercer Island)	1981	840	B.t. & trapping	Infested	236 moths in '81.
Clark County, WA	1981	20	carbaryl	Infested	Moths on edge of treatment block.
" " "	1982	120	acephate (2)	Infested	
" " "	1983	360	B.t. (3)	Eradicated	No moths trapped in '83 or '84.

Marion County, OR (Salem)	1982	3,976	carbaryl (2)	Infested	7 moths trapped in '82.
" " "	1983		mass trapping	---	10 moths trapped in '83.
" " "	1984			Eradicated	No moths trapped in '84.
McHenry County, IL (Crystal Lake)	1982	100	carbaryl (2)	Eradicated	Remains uninfested.
Lake County, IL (Lake Zurich)	1982	80	carbaryl (1)	Eradicated	Remains uninfested.
Lake County, IL (Lindenhurst)	1982	50	carbaryl (2)	Eradicated	" "
Lake County, IL (Lake Forest)	1982	10	carbaryl (2)	Eradicated	" "
Tazewell County, IL (Morton)	1982	5	carbaryl (2)	Eradicated	" "
Vigo County, IN (Terre Haute)	1982	500	carbaryl (2)	Eradicated	" "
Grand Traverse County, MI (Peninsula Twp.)	1982	728	carbaryl (2)	Eradicated	" "
Muskegon County, MI (Roosevelt Park)	1982	653	carbaryl (2)	Eradicated	" "
Ottawa County, MI (Grand Haven Twp.)	1982	550	carbaryl (2)	Eradicated	Remains Uninfested.
Belmont County, OH (Barkcamp State Park)	1982	120	carbaryl (2)	Eradicated	" "
Hamilton County OH (Anderson Twp.)	1982	146	carbaryl (2)	Eradicated	" "
Hamilton County, OH (Paul Meadows)	1982	38	carbaryl (2)	Eradicated	" "
Hamilton County, OH Montgomery)	1982	38	carbaryl (2)	Eradicated	" "
Cuyahoga County, OH (Solon)	1982	54	carbaryl (2)	Eradicated	" "
Cuyahoga County, OH (Pepper Pike)	1982	87	carbaryl (2)	Eradicated	" "
Cuyahoga County, OH (Bay Village)	1982	38	carbaryl (2)	Eradicated	" "
Tuscarawas County, OH (Newcomertown)	1982	93	carbaryl (2)	Eradicated	" "
Stark County, OH (Uniontown)	1982	58	carbaryl (2)	Eradicated	" "
Portage County, OH (Freedom Twp.)	1982	57	carbaryl (2)	Eradicated	" "
Lucas County, OH (Ottawa Hills)	1982	213	carbaryl (2)	Eradicated	" "
Johnson County, NC (Selma-KOA CG)	1982	100	Dimilin (2)	Eradicated	" "
Wake County, NC (Raleigh)	1982	225	B.t. (2)	Eradicated	" "
Fulton & Sharp Counties, AK (Hardy)	1982	1,526	carbaryl (2)	Eradicated	" "
Mobile County, AL	1982	5	carbaryl (2)	Eradicated	" "
Santa Barbara County, CA	1982	9,600	B.t.	Eradicated	" "
" " " "	1982	600	carbaryl		
Horry County, SC (Windjammer Village)	1982	100	sterile males	Eradicated	No moths trapped in '84.
Dane County, WI (Monona)	1982	300	mass trapping	Infested	114 moths trapped in '82.
" " "	1983	300	" "	Infested	40 moths trapped in '83.
" " "	1984	120	" "	Infested	7 moths trapped in '84.
Alameda County, CA (Pleasanton)	1983	160	carbaryl (3)	Eradicated	
Contra Costa County, CA (Clayton)	1983	64	carbaryl (3)	Eradicated	
Los Angelos County, CA (Westlake Village)	1983	100	carbaryl (3)	Eradicated	
Marin County, CA (Novato)	1983	10	carbaryl (3)	Eradicated	
San Mateo County, CA (Fair Oaks)	1983	40	carbaryl (3)	Eradicated	
San Mateo County, CA (San Mateo)	1983	50	carbaryl (3)	Eradicated	
Santa Clara County, CA (Campbell)	1983	160	carbaryl (3)	Eradicated	
Santa Clara County, CA (Palo Alto)	1983	160	carbaryl (3)	Eradicated	
Santa Clara County, CA (Los Altos)	1983	200	carbaryl (3)	Eradicated	

DuPage County, IL (Downers Grove)	1982	800	B.t. (2)	Infested	
" " " "	1983	50	B.t. (2)	"	1 moth trapped in treatment blocks in '83.
" " " "	1983	367	mass trapping		
" " " "	1984	40	mass trapping	Eradicated	No moths trapped in '84.
DuPage County, IL (Naperville)	1982	60	B.t. (2)		
" " " "	1983	40	B.t. (2)	Eradicated	
" " " "	1983	200	mass trapping		
DuPage County, IL (Wood Dale/Bensonville)	1982	500	B.t. (2)		
" " " " "	1983	989	B.t. (2)	Infested	13 moths trapped in treatment blocks in '83.
" " " " "	1983	1,532	mass trapping		
" " " " "	1984	643	mass trapping	Eradicated	No moths trapped in '84.
Kane County, IL (St. Charles)	1983	90	B.t. (3)	Infested	1 moth trapped in treatment block in '83.
" " " "	1984	60	mass trapping	Eradicated	No moths trapped in '84.
Elkhart County, IN (Goshen)	1983	80	carbaryl (2)	Eradicated	Only 80 acres of 300 acres treated. Moths trapped on periphery of area.
Kalamazoo County, MI (Portage Twp.)	1983	800	carbaryl (2)	Eradicated	No moths trapped in '83.
Muskegon County, MI (Norton Shores Twp.)	1983	160	carbaryl (2)	Eradicated	" " " " "
Van Buren County, MI (Antwerp Twp.)	1983	400	carbaryl (2)	Eradicated	" " " " "
Ramsey County, MN (St. Paul)	1983	300	carbaryl (2)	Eradicated	No moths trapped in treatment area in '83.
Washington County, MN (Woodbury)	1983	130	carbaryl (2)	Eradicated	" " " " "
Carteret County, NC (Beaufort)	1983	170	disparlure	--	Evaluate in 1984.
" " " "	1984	170	B.t. & mass trapping	Infested	14 moths trapped in '84.
Franklin County, OH (Blacklick)	1983	120	carbaryl (2)	Eradicated	No moths trapped in '83.
Franklin County, OH (Jefferson Twp.)	1983	80	carbaryl (2)	Eradicated	" " " " "
Hamilton County, OH (Cincinnati)	1983	105	carbaryl (2)	Eradicated	" " " " "
Hamilton County, OH (Anderson Twp.)	1983	130	carbaryl (2)	Eradicated	" " " " "
Knox County, OH (Mt. Vernon)	1983	65	carbaryl (2)	Eradicated	" " " " "
Lucas County, OH (Sylvania Twp.)	1983	160	carbaryl (2)	Infested	
" " " " "	1984	106	carbaryl (2)	Eradicated	No moths trapped in '84.
Marion County, OR (Salem)	1983	50	carbaryl (2)	Eradicated	No moths trapped in '83.
Jasper County, OH (Americana CG)	1983	40	carbaryl (3)	Eradicated	No moths trapped in '83.
Horry County, SC (Ocean Lakes CG)	1983	100	carbaryl (3)	Infested	49 moths trapped in '83.
" " " " " "	1984	100	mass trapping	Infested	14 moths trapped in '84.
Floyd County, VA (Tuggles Gap)	1983	4,001	disparlure	Eradicated	No moths trapped in treatment block/entire county in '83. No moths trapped in '84.

King County, WA (Ravenna Park)	1983	1,040	B.t. (2) & mass trapping	Infested	32 moths trapped in '83.
" " " " "	1984	3,586	B.t. (3)	Infested	7 moths trapped in '84.
Pierce County, WA (Tacoma East)	1983	800	B.t. (2) & mass trapping	Infested	5 moths trapped in '83.
" " " " "	1984	--	--	Eradicated	No moths trapped in '84.
Pierce County, WA (Tacoma West)	1983	320	B.t. (2) & mass trapping	Infested	33 moths trapped in '83.
" " " " "	1984	2,254	B.t. (3) & Orthene	Infested	6 moths trapped in '84.
		<5			
Waukesha County, WI (Elm Grove)	1983	60	B.t. (2) & mass trapping	Infested	3 moths trapped in '83.
" " " " "	1984	230	mass trapping	Eradicated	No moths trapped in '84.
Alameda County, CA (Livermore)	1984	165	carbaryl (3)	Eradicated	No moths trapped in '84.
" " " (Oakland)	1984	170	carbaryl (3)	Eradicated	" " " "
Santa Clara County, CA (San Jose)	1984	220	carbaryl (3)	Eradicated	" " " " "
San Diego County, CA (San Diego)	1984	90	carbaryl (3)	Eradicated	" " " " "
" " " " "	1984	115	B.t. (6)	--	1 moth trapped in '84.
Kane County, IL (Aurora)	1984	4	B.t. (2) & mass trapping	Eradicated	
" " " "	1984	84			
Lake County, IL (Mundelein)	1984	3	B.t. (2) & mass trapping	Eradicated	No moths trapped in '84.
" " " "	1984	84		"	" " " " "
Lake County, IL (Kildeer)	1984	86	mass trapping	Infested	3 moths trapped in '84.
Kane County, IL (Geneva)	1984	40	mass trapping	Infested	10 moths trapped in '84.
Bartholomew County, IN (Columbus)	1984	80	mass trapping	Eradicated	No moths trapped in '84.
Marion County, IN (Indianapolis--Site 1)	1984	160	mass trapping	Infested	7 moths trapped in '84.
" " " " Site 2)	1984	160	mass trapping	Eradicated	No moths trapped in '84.
Eaton County, MI (Charlotte)	1984	217	carbaryl (2)	--	Moths trapped outside treatment area in '84. Part of area generally infested.
Menominee County, MI (Spalding Twp.)	1984	306	carbaryl (2)	Eradicated	No moths trapped in '84.
Benton County, MN (Sauk Rapids)	1984	20	B.t. (2) & mass trapping	Eradicated	No moths trapped in '84.
Hennepin County, MN (St. Anthony)	1984	30	B.t. (2) & mass trapping	Eradicated	No moths trapped in '84.
Washington County, MN (Stillwater)	1984	32	B.t. (2) &		
" " " "	"	90	mass trapping	Eradicated	No moths trapped in '84.
Franklin County, OH (Little Turtle)	1984	50	carbaryl (2)	Eradicated	No moths trapped in '84.
Lucas County, OH (Sylvania)	1984	106	carbaryl (2)	Eradicated	No moths trapped in '84.
Benton County, OR (Corvallis area)	1984	960	B.t. (3) & mass trapping	--	No moths trapped in 2 blocks. 1 moth trapped in 1 block.

Marion County, OR (Salem area)	1984	8,470	B.t. (3) & mass trapping		No moths trapped in 8 blocks; 12 moths trapped in 3 blocks.
Multnomah County, OR (Portland area)	1984	2,155	B.t. (3) & mass trapping	--	No moths trapped in 1 block; 11 moths trapped in 2 blocks.
" " " " "	1984	85	mass trapping	--	2 moths trapped in one trap.
" " " (Gresham)	1984	320	B.t. (3) & mass trapping	Eradicated	No moths caught in '84.
Watauga County, NC	1984	615	Dimilin (2)	--	1 moth trapped in '84.
" " "	1984	611	B.t. (2)	--	No moths trapped in '84.
Johnson County, TN	1984	13,039	Dimilin (2)	Infested	61 moths trapped in '84.
" " "	1984	1,178	B.t. (2)	Infested	5 moths trapped in '84
Montgomery County, VA	1984	1,200	Dimilin (2)	--	2 moths trapped in '84.
" " "	1984	320	Luretape		
Patrick County, VA	1984	525	Dimilin (2)	--	1 moth trapped in '84.
" " "	1984	40	Luretape		
King County, WA (Medina)	1984	80	B.t. (3)	Infested	2 moths trapped in 84.
Snohomish County, WA	1984	1,484	B.t. (3)	--	1 moth trapped in '84.
" " "	1984	<1	Orthene		

APPENDIX F

ANALYSIS OF HUMAN HEALTH RISKS OF USING ACEPHATE, CARBARYL, DIFLUBENZURON, AND TRICHLORFON INSECTICIDES IN GYPSY MOTH SUPPRESSION AND ERADICATION PROJECTS

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PURPOSE AND SCOPE

The purpose of this risk analysis is to provide an evaluation of human health risks associated with using four chemical insecticides: acephate, carbaryl, diflubenzuron, and trichlorfon. These pesticides are used to suppress or eradicate the gypsy moth. This analysis was designed for both the decisionmaker and the interested public. It includes a discussion of risks during normal treatment operations and risks arising from accidents and other abnormal situations.

Exposures and potential health risks to the public living in and near areas to be treated have been estimated for projects involving eradication and suppression. Occupational exposures to mixer/loaders, and other project personnel and the relation of these exposures to possible health effects were also determined. These are shown separately from exposures to which the general public might be subjected.

Most abnormal exposure situations are short term (acute), but may result in greater than usual exposure levels to people--project workers and the public alike. Proper safety measures will minimize any deleterious effects posed by these situations. However, estimates of frequency of occurrence based upon historic experience and postulated scenarios are calculated for these abnormal situations in order to put their health risks into perspective.

In conducting the risk analysis, some uncertainties or data gaps were encountered (such as specific exposure data, extrapolation from animal tests to humans, and the question of the carcinogenic potential of trichlorfon or the possibility of N-nitrosocarbaryl being formed and causing cancer). In such cases, the uncertainties or data gaps are identified. In most cases, these data gaps were filled by extrapolation from the existing data base. Where the existing data base was not sufficient, assumptions were used to model exposure levels. When

assumptions were necessary, a range of assumptions from realistic to extreme (worst case) were used. In all cases, the extrapolations made and the assumptions used in this analysis were designed to lead to conserative estimates. It may be argued that the doses represented as being realistic in this analysis are really worst case.

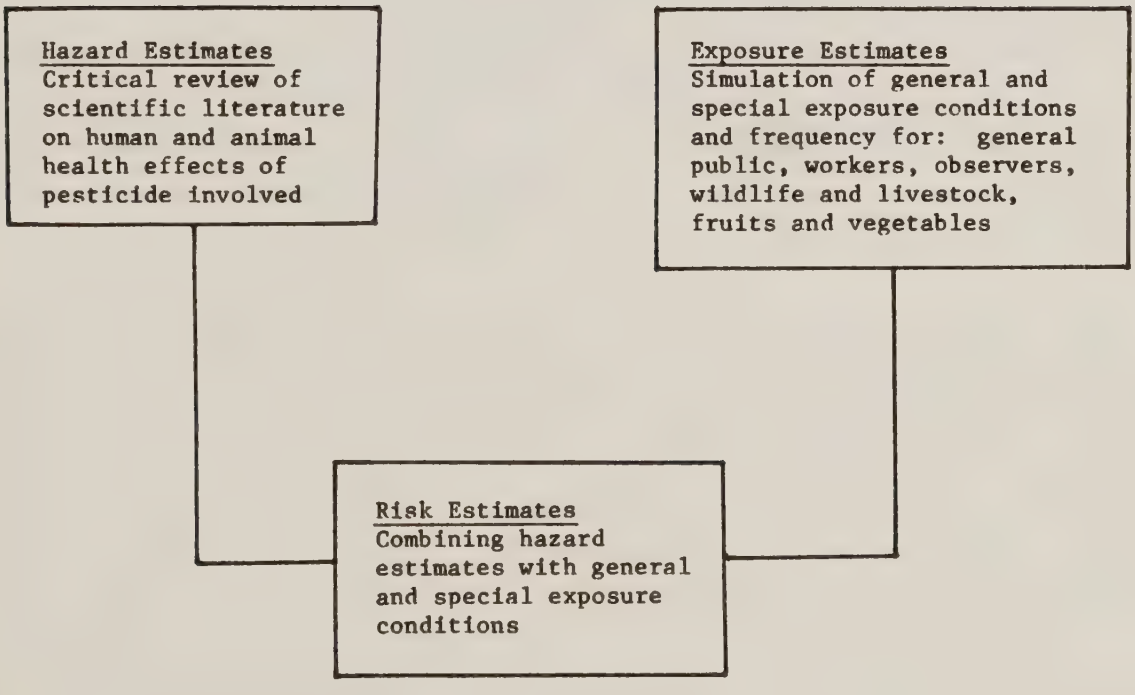
The rationale and basis for the assumptions, and all calculations associated with the assumptions, are included to provide readers with adequate information to make a reasoned choice among alternatives. In assessing the available information, no data gap or scientific uncertainty encountered was so significant that the risk analysis could not be completed.

RATIONALE FOR THE RISK ANALYSIS

The methodology used in this risk analysis consists of three individual components: 1) hazard identification through a review of the toxicological data for each of the specific insecticides; 2) exposure analysis which includes the probable amounts of exposure to humans, frequency of exposure, and the number of people possibly exposed; and 3) evaluation of risk by comparing the results from the toxicological review to the data on human exposure in order to calculate the risk to an individual or to society.

To aid the reader in following the steps and logic of the risk analysis, a flow chart has been included on the following page that presents the relationship of the three components discussed above. Additional flow charts for Hazard Estimates, Exposure Estimates, and Risk Estimates preceed the specific sections in the analysis that deal with those components. For example, the flow chart for Hazard Estimates preceeds the section titled, Review of Toxicological Studies. The flow charts are included to give the reader a quick review of what will be presented in the subsequent section, and to provide a graphical representation of the logic of the analysis.

PROCEDURE FOR RISK ASSESSMENT



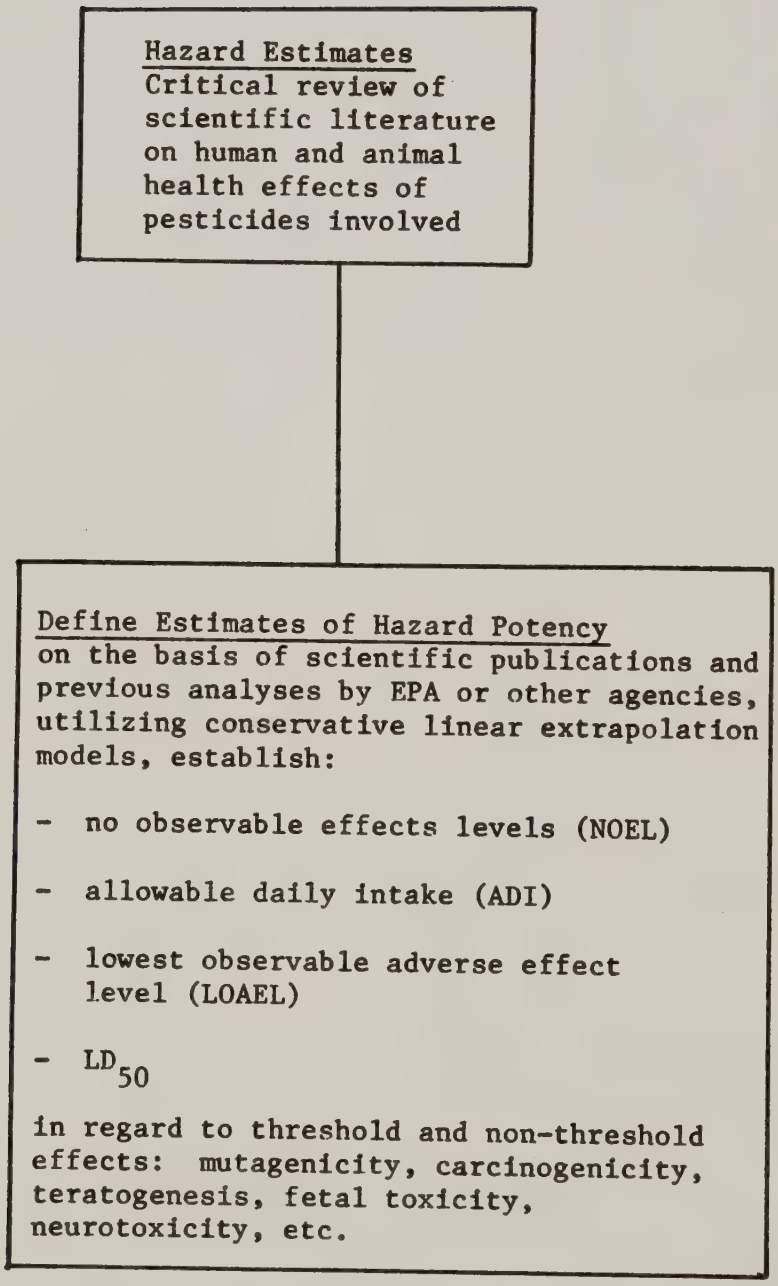
The toxicological data base of each insecticide was reviewed for acute and chronic effects on test animals. The No Observable Effect Levels (NOEL), Acceptable Daily Intake (ADI), and where available, the Lowest Observable Adverse Effect Level (LOAEL) were obtained from the literature or from the U.S. Environmental Protection Agency (EPA). These were used to evaluate threshold responses such as teratogenicity (birth defects), fetotoxicity (effect on the fetus), and neurotoxicity (effect on the nervous system). For single, short-term exposures to large doses, as would result from accidents, the LD_{50} dose was used as an indication of acute dose response. Cancer and mutational outcomes were evaluated differently. A cancer risk analysis was conducted based upon average daily exposure over a 70-year lifetime. A cancer risk analysis was conducted on trichlorfon, because it is mutagenic in experimental animals and therefore a suspect carcinogen. A cancer risk analysis was also conducted on N-nitrosocarbaryl, a proven carcinogen and a nitrosation product of carbaryl which could possibly form as a result of carbaryl application. The risks of heritable mutations were discussed based on available test data on bacteria, yeast, plants, mammalian cells in culture, and whole animal studies.

This risk analysis is based on both eradication and suppression project scenarios in urban, suburban, and rural areas. A time span of 70 years was used as the average lifetime of an individual. The frequency of applications over a 70-year lifetime is identified for each type of project. This permits a determination of insecticide exposure during a lifetime to all possible recipients.

The estimate of exposure is based upon a 1 lb. active ingredient (a.i.) per acre application. This exposure provides the basis for estimating the doses to people via inhalation, ingestion, and dermal pathways. Each exposure pathway results in a single dose to an individual. The sum of all exposures provides the estimate of the maximum dose from a 1 pound per acre application rate. The maximum dose levels thus computed are multiplied by the highest recommended application rate for each of the four insecticides

to determine specific doses. The specific doses are scaled upward to account for mixing and application errors, and are identified as realistic and worst case doses. Exposures resulting from airplane and truck accidents are treated in a similar manner; however, the affected population is much smaller and the exposure pathways are different.

Maximum lifetime doses to an individual are calculated for a range of possible exposure scenarios. These scenarios may result from an individual contacting insecticides via the application process, the consumption of contaminated food and water, or a combination of these. The calculated doses form the basis for evaluating the risk to the worker, to the general public, and to sensitive individuals when compared to thresholds established from animal studies. To provide a perspective in viewing risks from the use of these insecticides, the doses are compared to everyday risks which occur in our society.


Hazard Estimates
Critical review of scientific literature on human and animal health effects of pesticides involved
Define Estimates of Hazard Potency
on the basis of scientific publications and previous analyses by EPA or other agencies, utilizing conservative linear extrapolation models, establish:
- no observable effects levels (NOEL)
- allowable daily intake (ADI)
- lowest observable adverse effect level (LOAEL)
- LD_{50}
in regard to threshold and non-threshold effects: mutagenicity, carcinogenicity, teratogenesis, fetal toxicity, neurotoxicity, etc.

REVIEW OF TOXICOLOGICAL STUDIES

Background Information

Because of major issues and concerns raised during the scoping process and Oregon Environmental Council, et al. vs. Leonard Kunzman, et al., the literature review concentrated on studies dealing with birth defects (teratogenicity, embryotoxicity, and fetotoxicity), mutagenicity, and carcinogenicity for the four chemical insecticides. The review of scientific literature included searches of Chemical Abstracts, Biological Abstracts, Bibliography of Agriculture, Commonwealth Agriculture Bureau, Life Sciences, Med-line, Excerpta Medica, Aquatic Sciences Abstracts, Enviroline, Environmental Bibliography, and Pollution Abstracts. Requests for specific information on environmental fate, toxicologies and reviews were made to the individual registrants and EPA. The data requirements for pesticide registration proposed by the EPA (USEPA 1984c) were used as a guide to identify appropriate studies for evaluating possible human health impacts. In addition, the National Institute for Occupational Safety and Health reviews the same type of information in setting permissible exposure limits for workers, as does EPA and the WHO (World Health Organization) in setting acceptable daily intakes (ADI) for the general public. In reviewing the available toxicological data, emphasis was placed on studies that involve oral, dermal or inhalation exposure routes, rather than those involving injected doses, since the former studies are more relevant to possible exposure from the aerial application of insecticides.

The use of data from refereed journals was meant to reduce scientific uncertainty about the validity of the information used for initial pesticide registration. The validity of some data submitted to the EPA has been questioned because of falsified data provided by Industrial Bio-Test Laboratories (IBT) to support certain pesticide registrations. Acephate was the only insecticide in this analysis that had a substantial number of toxicity tests (19) performed by IBT and which were used to support registration. All of the tests on acephate have either been replaced, validated, or judged unnecessary to support registration (USEPA 1983b).

Neither trichlorfon nor diflubenzuron was tested by IBT, while carbaryl had one test. No IBT data judged by EPA to be invalid were used in the development of this risk analysis.

All the insecticides proposed for use in gypsy moth suppression or eradication projects are currently registered by the EPA for the control of gypsy moth larvae under the Federal Insecticide, Fungicide and Rodenticide Act (FIFRA) as amended. Registrations are either conditional or unconditional based on data requirements set forth in sections 3(c)(5) or 3(c)(7) of FIFRA. A conditional registration means that the pesticide has been granted registration status by EPA prior to meeting all the data requirements set forth in Section 3(c)(5) (see USEPA 1984c). A pesticide can be granted conditional registration only if existing studies or those in progress indicate that it will not significantly increase the risk of unreasonable adverse effects on the environment (USEPA 1983c). This analysis does not differentiate between conditional or unconditional registrations and furthermore is independent of the registration process.

Since the issuance of the 1984 FEIS (Final Environmental Impact Statement, USDA FS/APHIS 1984), EPA has issued registration standards for carbaryl and trichlorfon (USEPA 1984 a,b) and is in the process of reviewing acephate. EPA's registration standards program was established to comply with the congressional mandate that EPA reregister all pesticides. The program involves a thorough review of the scientific data base supporting pesticide registration. This review identifies essential missing data which may not have been required when the pesticide was initially registered. It also identifies studies that are considered insufficient by today's new study protocol standards. The registration standards program also provides the basis for EPA to require registrants to submit the missing or replacement data (data call-in) under the authority of Section 3(c)(2) B of FIFRA.

The registrants of carbaryl have been requested to repeat the teratology (birth defect) study in beagle dogs and to conduct a metabolism study in beagle dogs versus monkeys or rats. They were also required to conduct a 1-year feeding study in dogs to determine the effects of carbaryl on kidney function, and additional studies on environmental fate and impact to nontarget organisms. All labels for end-use products must bear an

Environmental Hazards paragraph designed to protect wildlife (USEPA 1984a). Data call-in for trichlorfon is much more extensive and includes acute toxicity studies on inhalation, eye and dermal sensitization, subchronic and chronic feeding and oncogenicity studies (because available studies have deficiencies in protocol design or reporting). Also required are: mutagenicity studies to determine possible chromosome aberrations; a 1-year feeding study in dogs to support an ADI; a dietary teratology study; and studies to identify residue components in specific commodities so that the adequacy of existing food tolerances can be fully assessed.

Threshold Responses

The main purpose of toxicity tests in animals is to provide data that can be used to assess the risk or evaluate the hazard associated with the use of a particular substance. The characteristics of exposure and the varying toxicological effects come together in a correlative relationship customarily referred to as the dose-response relationship (see, for example, Doull et al. 1980). Nonlinear dose-response curves can be prepared for threshold effects. The term "threshold" is used to describe the dividing line between the no-effect and effect levels of exposure. The time-honored approach for establishing safe levels of pesticides to which humans may be exposed is to divide the threshold dose or NOEL established from chronic animal studies by a "safety factor" (Doull et al. 1980 and NAS-NRC 1977). The safety factors are needed to account for differences in route and duration of exposure, absorption, metabolism, and excretion between humans and test animals. For example, on a body-weight basis, man is generally more vulnerable to drugs than experimental animals by a factor of 6-12 (NAS-NRC 1977). If dose is scaled on a surface area basis, this increased vulnerability disappears. ADIs are established by EPA or WHO by dividing a NOEL, usually from a multigeneration feeding study, by a safety factor that takes into consideration both intraspecies and interspecies differences.

NOELs (no observable effect levels) from various animal experiments identified for the four insecticides are summarized in Tables 1-4. In studies where no effect was observed at any dose, the highest value tested

is identified as the NOEL. ADIs established by either EPA or WHO are listed in Table 7. With the exception of trichlorfon, ADI values established by EPA were used in this analysis. Since EPA concluded that their ADI for trichlorfon (0.125 mg/kg/day) was based on inadequate data (USEPA 1984b), the lower ADI established by WHO was used in its place. The information summarized in the Tables indicates that the insecticides pose some potential for causing adverse human health effects: acute toxicity, chronic cholinesterase depressions, birth defects, or systemic toxicity if specific dose levels (the NOELs or ADIs) are exceeded. It is recognized that developmental toxicology includes many adverse effects including terotogenicity, fetotoxicity, embryotoxicity, altered growth and malformations. Since at present there are no generally acceptable models for determining risk of developmental toxicity, these responses were analyzed as if thresholds existed (USEPA 1984k).

The characteristic of diflubenzuron to cause methemoglobin (MHb) and sulfhemoglobin (SHb) formation has generated some concern that exposure might result in significant impairment of oxygen transport capacity. The FAO (1982) has reviewed proprietary and other published data on the potential for diflubenzuron to cause methemoglobinemia. Groups of 10 mice were given 8, 40, 200, 1,000, and 5,000 mg of diflubenzuron per kg daily for 14 days. No effect was detected at 200 mg/kg/day and below. An experiment with rats given 5000 mg/kg/day for 8 days disclosed "marginal" MHb and SHb from the first day. The specific data were not provided. Rabbits fed a diet containing 640 ppm of diflubenzuron for 21 days were found to have increased SHb for 5 hours onward, while MHb did not increase until the 5th day. Recovery was complete 2 weeks after the end of the experiment. In female cats, diflubenzuron caused a dose related increase in MHb at all doses between 30 and 100 mg/kg/day for 21 days. Males showed no effect at 70 mg/kg/day. The maximum effect was 11.8 percent. SHb was seen in all groups.

In experimental animals, while there is not a statistically significant difference between the effect of chronic doses of about 1.5 mg/kg/day and about 7 mg/kg/day, the dose response curve for rodent lifetime experiments shows the latter intake to be clearly part of an orderly dose related

increase with values on the order of 1.5 to 2 percent MHb. A dose of 1.5 mg/kg/day certainly has no effect in mice and in rats 1 mg/kg would be indistinguishable from no treatment. EPA has set a NOEL of 1.1 mg/kg/day for MHb and SHb (USEPA 1984).

MHb is a chemically oxidized form of hemoglobin with iron in Fe^{+3} rather than Fe^{+2} state. It is therefore useless in the transport of oxygen. It is produced naturally and continuously in all mammals; the true oxidation by molecular oxygen occurs in the absence of any extraneous chemicals. All humans have up to about 1 percent hemoglobin in this form at all times and smokers will have substantially greater amounts (Dr. Frank Dost, Oregon State University, Personal Communication).

All mammals have in the red blood cells a MHb reductase (cytochrome b5 reductase) system that maintains hemoglobin in the reduced active form. The system is less well developed in the newborn and is genetically deficient in a few individuals. Sulfhemoglobin also occurs in normal organisms and is often increased in company with increased MHb of chemical origin. It cannot be reduced by the MHb reductase system but does slowly revert spontaneously to hemoglobin. It is also removed in the normal process of destruction and replacement of red blood cells.

The biochemistry of sulfhemoglobin has received almost no attention. The last three cumulative indices to Chemical Abstracts list only seven references, of which three shed some speculative light on the mechanism of sulfhemoglobin formation. While there is no firm evidence, it seems that the formation of sulfhemoglobin may depend on the initial oxidation step that also leads to methemoglobin formation, and is dependent also on availability of sulfhydryl containing biological molecules. With respect to the problem of exposure to diflubenzuron, sulfhemoglobin formation does not exceed that of methemoglobin (Dr. Frank Dost, Personal Communication).

Nonthreshold Responses

To evaluate the ability of the insecticides to produce genotoxic effects such as tumor initiation or heritable mutations, NOELs or thresholds doses were not used. This was done because it is conceivable that only one or a few molecules of an active chemical may be sufficient to cause certain types of changes in DNA which could result in either the formation of neoplastically transformed cells or heritable mutagenic effects. In the case of cancer, individual and population risks can be quantified using various models if there is scientific evidence to suggest a chemical is a carcinogen. Since quantitative risk models are not available for mutagenicity, a multi-step process of evaluating a pesticide's ability to cause mutations and to interact with germinal cells is used to assess the qualitative potential of mutagenic risk in humans (see, for example, USEPA 1984c, or USEPA 1980c). The first step involves an analysis of the evidence of a pesticide's ability to cause mutations in bacteria, microorganisms, insects, plants, mammalian cells in culture and germinal cells in whole animals; while the second step involves an analysis of its ability to produce these events in mammalian gonads. Greater weight is placed on tests that show changes in germinal tissues than in somatic cells, on tests performed in vivo (within the body) rather than in vitro (outside the body), and in mammalian species rather than in submammalian species (USEPA 1984j). Table 18, provided by Dr. David Brusick with Litton Bionetics, Inc., presents a listing of various tests and their value in predicting a chemical's mammalian carcinogenic and heritable mutagenic potential.

Mutagenicity.--Diflubenzuron was found to be nonmutagenic even at high dose levels (MacGregor et al. 1979; and USEPA 1984d) in Ames Salmonella reverse mutation tests, the micronucleus test in mice, and the mouse lymphoma forward mutation assay. EPA reviewed existing data on mutagenicity of carbaryl during the Rebuttable Presumption Against Registration (RPAR) process (USEPA 1980a) and found that carbaryl was reported to produce gene mutations in bacteria, fruit flies (Drosophila) and mammalian cells in culture. Cytogenic tests indicate that carbaryl may induce chromosomal effects in mammalian cells in culture, whole animals,

and plants, and cause primary DNA damage in cultured human cells (USEPA 1980a and USEPA 1984e). There is also some suggestive but not conclusive evidence that carbaryl may reach mammalian gonads. This includes two epidemiological studies dealing with sperm counts of workers formulating carbaryl, and a number of gonadal studies in rodents. However, the epidemiological studies are flawed because the differential age of the workers could account for the elevated sperm abnormalities and there were no differences between low or high exposure. The study also showed that workers, who were no longer exposed to carbaryl, did not have elevated sperm abnormalities. This type of information adds to the high uncertainty about the mutagenic risk of carbaryl to humans. EPA concluded that the weight of evidence is that carbaryl poses low mutagenic risk due to the weak mutagenic response noted in the mutagenicity studies and due to the suggestive rather than conclusive evidence about whether carbaryl reaches germinal tissue.

Acephate has been reported to be weakly mutagenic in studies with Salmonella typhimurium, Escherichia coli, and Saccharomyces cerevisiae D7 yeast (Simmon 1979, and Jones et al. 1984, and USEPA 1984f). DNA repair assays were found to be negative in bacteria, positive with yeast, and weakly positive in mammalian cells in culture without metabolic activation, but negative with activation. A sister chromatid exchange assay in Chinese hamster ovary cells in culture was very positive (Jones et al. 1984 and USEPA 1984f). However, sister chromatid exchange and chromosome aberration tests in mice and monkeys were negative for acephate (Jones et al. 1984 and USEPA 1984f). A dominant lethal test in mice showed no effect at the highest dose tested (1000 ppm). The overall results of these mutagenicity studies indicate that acephate can induce gene mutations, DNA repair, and sister chromatid exchanges in submammalian or mammalian cells in culture. However, studies in whole mammals indicate that these effects, in addition to structural chromosome aberrations, are not produced at detectable levels in doses that can be tolerated by intact mammalian systems.

Trichlorfon increased reverse mutations in S. typhimurium, E. coli, and yeast (S. cerevisiae) (Simmon 1979; Jones et al. 1984). Concentration-related increases in the sister chromatid exchange in Chinese

hamster ovary cells in culture with and without metabolic activations were also reported by Jones et al. (1984) for trichlorfon. The same authors also report concentration related increases in mutagenic frequency in mouse lymphoma cells, both with and without metabolic activation. Trichlorfon did not cause chromosome damage in the one in vivo study (mouse micronucleus test) reviewed by Jones et al. (1984) at doses ranging from 100 to 400 mg/kg. Degraeve et al. (1981) also reported negative cytogenetic effects in bone marrow and spermatogonia in mice at dosages up to 100 mg/kg. However, Kiraly et al. (1977) found an increased frequency of chromatid-type aberrations in workers who manufactured trichlorfon. These overall results indicate that trichlorfon is mutagenic in bacteria, yeast and mammalian cells in culture. Whole animal studies are inconclusive because of the inconsistency in results. However, there is an indication that some risk of heritable mutation might exist from exposure to trichlorfon.

Cancer.--It was concluded from reviewing the data available that acephate (USEPA 1982a, 1984f), carbaryl (USEPA 1980a, 1984a), and diflubenzuron (Quarles 1980; Patel and Santolucito 1980) are not carcinogenic and therefore do not pose a cancer risk. However, when diflubenzuron was first registered, EPA's Carcinogen Assessment Group reported suggestive evidence of carcinogenic response to diflubenzuron in female mice. Because of several deficiencies in the mouse cancer study, a new carcinogenicity study was required as a condition of the registration granted in 1976. The study has been completed and reviewed by EPA (USEPA 1984h and 1984i). The new oncogencity studies, conducted on mice and rats, show that diflubenzuron is noncarcinogenic at all doses tested.

Although carbaryl itself was not found to be carcinogenic, there is some uncertainty about the ability of carbaryl to combine with nitrite ions in nature to form N-nitrosocarbaryl, a compound which has a demonstrated carcinogenic potential (see FEIS Appendix C and p. 56-57). This is of concern because the nitrosation of secondary and tertiary amines (to form nitrosamines) is a common process in acidic environments (see, for example, Fan and Tannenbaum 1973a). However, numerous studies (Fan and Tannenbaum 1973b; Fiddler et al. 1973) have indicated that ascorbic acid (Vitamin C)

and Vitamin E will effectively block the synthesis of nitrosamines from the interaction of secondary amines with nitrite at a pH of 3-5, normally found in the stomach. The pH of stomach contents following a meal is slowly lowered from 5 to 1. At a pH of 1.5, N-nitrosocarbaryl is rapidly denitrosated to regenerate carbaryl (Rickard and Dorough 1979), which is noncarcinogenic. However, the reaction conditions, kinetics, and yields from laboratory studies of N-nitrosocarbaryl permit an analysis of the risk to humans exposed to carbaryl, if we make a worst case assumption that N-nitrosocarbaryl does form in the human stomach.

There is uncertainty about acephate and trichlorfon as potential carcinogens because of their mutagenic potential and the relationship between mutagenicity and cancer (Meselson and Russell 1977; McCann and Ames 1977). Preussmann (1968) also indicated that trichlorfon may be a weak carcinogen because of its alkylating activity. Also, the Registration Standard stated that it may cause cancer at 500 to 1,000 ppm in the diet of test animals (USEPA 1984b). However, EPA concluded that available data were inadequate for them to do a quantitative risk assessment for oncogenicity. However, studies by Teichmann and Hauschild (1978), Teichmann and Schmidt (1978), Teichmann et al. (1978), and Machemer (1981) showed no statistically significant differences in malignant and benign tumors between untreated animals and those treated with trichlorfon doses as high as 1,000 ppm (in diet). Because of this uncertainty about the cancer potential of trichlorfon, and because of its documented mutagenic potential, trichlorfon was assumed to be a carcinogen and analyzed as such.

Although information available from published sources (USEPA 1982a and 1984f) indicated that acephate had no carcinogenic potential, the comment letter to the draft supplement to the FEIS provided by EPA (See Appendix G, letter 29) points out that there is some indication of potential oncogenic activity for acephate. This carcinogenic potential is currently under evaluation by EPA.

Since no carcinogenic potency values for N-nitrosocarbaryl, trichlorfon, or acephate were available in published literature, they were determined using a simple linear model for cancer incidence modified from Crouch and Wilson (1979) as follows:

$$R = \alpha + \beta d$$

Where:

R = the risk of incidence of cancer
α = the spontaneous incident rate; (incidence in untreated control)
β = the cancer potency
d = the lifetime dose of the test animal in mg/kg/day

It is assumed that a substance that induces a carcinogenic response in animals has the capacity to cause cancer in humans. Thus, animal bioassay results from high doses can be used to measure the cancer potency (β) of a chemical in rodents. Human risk due to exposure to the same carcinogen is calculated by extrapolating the cancer potency found in animals to humans:

$$R \text{ (in humans)} = \beta \times K \times d$$

Where:

β = cancer potency in the test animal
d = lifetime dose for humans
K = an interspecies multiplicative factor

The multiplicative factor (K) is applied to account for differnces in sensitivities between species. This factor is intended to correct for such things as differences in metabolism between species and differences in different routes of exposure or food consumption. It is also important to note that cancer potencies are first determined from animals that have been bred for their sensitivity to express specific cancers. Potencies from

these sensitive animals are then extrapolated to humans to cover the possibility of all types of cancer. Obviously, there is considerable uncertainty involved with such extrapolations.

Following the suggestion of Mantel and Schneiderman (1975), mg/surface area is an equivalent dose between species. Therefore, it provides a first approximation for extrapolating between species. Since surface area is proportional to body weight to the 2/3 power (wt. $^{2/3}$), the multiplicative factor K of:

$$K = (\text{wt human}/\text{wt animal})^{1/3}$$

(Mantel and Schneiderman 1975) will adjust the cancer potencies (β) for differnces in surface area. This is the same multiplication factor used by EPA (Crouch and Wilson 1979).

If animal exposure information is not given in terms of mg/kg/day, it must be converted to mg/kg/day. For example, ppm in diet can be converted to mg/day as follows:

$$m\ (\text{mg/day}) = \text{ppm} \times F \times \underline{r}$$

Where:

F = the weight of the food consumed per day in kg and

$\underline{r}$ = the absorption fraction assumed to be 1.0 (a worst case assumption).

An empirically derived food factor, f = F/w, which is the fraction of species body weight that is consumed per day as food, is used to calculate F (US EPA 1980b):

Species	$\underline{w}$ (kg)	$\underline{f}$
Human	70	0.028
Rat	0.35	0.05
Mice	0.03	0.13

N-nitrosocarbaryl.--Carbaryl can be nitrosated to form N-nitrosocarbaryl under conditions simulating those of the human stomach (e.g., mild acid, 37°C, in the presence of nitrite). The reaction product has been shown to be a direct acting carcinogen. Eisenbrand et al. (1976) reported that 29 percent of the treated male Sprague-Dawley rats died of squamous cell carcinoma of the forestomach when dosed twice a week with 130 mg/kg of N-nitrosocarbaryl. The average lifetime dose was 5,000 mg/kg body weight and the average time to death of the affected animals was 167 days. Three of the untreated animals died of lymphosarcomas and leukemia. However, we assumed α (the spontaneous incident rate) to be zero because these tumors were different from those caused by the treatment.

The average daily dose (d) was calculated by dividing the average lifetime dose (5000 mg/kg) by the average time of exposure (167 days):

$$d = 5000 \text{ mg/kg} \div 167 \text{ days}$$

$$= 29.94 \text{ mg/kg/day}$$

The incidence of cancer (R) in the rats is calculated as follows:

$$R = \alpha + \beta d$$

Where:

$R = 0.29$ (Eisenbrand et al. 1976)
$d = 29.94$ mg/kg/day
$= 0$

The cancer potency (β) in rats is calculated as follows:

$$\beta(\text{rat}) = \frac{R - \alpha}{d}$$

$$= 0.29/29.94 \text{ mg/kg/day}$$

$$= 0.0097 \text{ (mg/kg/day)}^{-1}$$

The rat cancer potency (β) is multiplied by the 1/3 power of the ratio of human (70 kg) to animal weight (0.35 kg) to estimate a human cancer potency as follows:

$$\beta(\text{human}) = 0.0097\ (\text{mg/kg/day})^{-1} \times (70\ \text{kg}/0.35\ \text{kg})^{1/3}$$
$$= 0.057\ (\text{mg/kg/day})^{-1}$$

Trichlorfon.--The same type of calculation can be made for trichlorfon using the data reported by Machemer (1981): 4 percent of treated rats had malignant tumors after 24 months of daily dietary exposure at 1,000 ppm; 7.5 percent of the untreated group had malignant tumors which would be the spontaneous tumor rate (α in R = α + β d), but we are assuming the response curve goes through zero, therefore α = 0 (a worst case assumption).

Lifetime average dose (in mg/kg/day) = ppm x F/w
= ppm x f
= 1000 ppm x 0.05
= 50 mg/kg/day

The cancer potency (β) for rats is calculated as follows:

β(rat) = R/d
= 0.04/50 mg/kg/day
= 0.0008 $(\text{mg/kg/day})^{-1}$

To extrapolate this value to humans, β(rat) is multiplied by K as follows:

$$\beta(\text{human}) = 0.0008\ (\text{mg/kg/day})^{-1} \times (70\text{kg}/0.35\text{kg})^{1/3}$$
$$= 0.0047\ (\text{mg/kg/day})^{-1}$$

Acephate.--USDA discussed the possible carcinogenic potential with Chevron Chemical Co. (Dr. L.R. Stelzer, personal communication). A carcinogenicity study was conducted for Chevron Chemical Co, by International Research and Development Corporation (Mattawan, Mich.). Seventy-five male and female Charles River CD-1 mice were given 0, 50, 250

and 1000 ppm acephate in their diet over a period of 104 weeks. Total hyperplastic liver nodules and hepatocellular carcinomas and adenomas in female mice were 4.0 percent in control, 5.4 percent at 50 ppm, 0.0 percent at 250 ppm, and 40.0 percent at 1000 ppm. For males the incidence of lesions was 20.0 percent in control, 13.3 percent at 50 ppm, 10.7 percent at 250 ppm and 22.7 percent at 1000 ppm. Apparently, it is the acephate related increase in tumors at the 1000 ppm dose in females that is raising the question about carcinogenic potential. It is important to note that the 1000 ppm dose was equivalent to 167 mg/kg/day which is approximately one-half the LD_{50} for mice (361 mg/kg). As one might expect, mice at the 1000 ppm dose level exhibited signs of acute toxicity. For example, the average body weight for the females dosed at 1000 ppm were 28.6 percent lower than the average weight for the untreated control. It could be argued (OSTP 1984) that the 250 ppm dose which produced only a 14.3 percent reduction in body weight, should have been the maximum dose. In that case, the study would have been negative.

The question that needs to be resolved is whether the increased carcinogenic activity is a result of acephate or the stress to the test animals. Since neither the Forest Service nor APHIS is in a position to debate this academic question, the agencies have chosen to analyze the cancer risk associated with acephate based on the worst case risk associated with the 1000 ppm data in female mice. The cancer potency for mice is calculated as follows:

$$\text{Rate of tumors at 1000 ppm} = 0.40 = \alpha + \beta d$$

$$0.40 = 0.04 + \beta(167 \text{ mg/kg/day})$$

$$\beta(\text{mice}) = 0.0021 \text{ (mg/kg/day)}^{-1}$$

To extrapolate this cancer potency to humans, β(mice) is multiplied by K as follows:

$$\beta(\text{human}) = 0.0021 \text{ (mg/kg/day)}^{-1} \times (70 \text{ kg}/.042 \text{ kg})^{1/3}$$

$$= 0.025 \text{ (mg/kg/day)}^{-1}$$

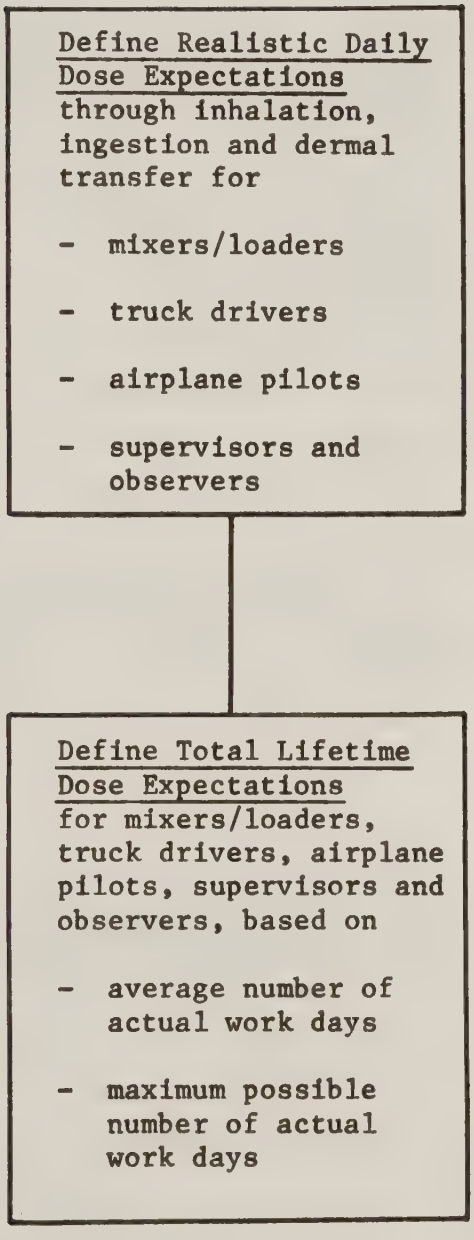
Diflubenzuron.--To complete the cancer risk analysis for the four insecticides, USDA discussed the recently completed oncogenicity studies on diflubenzuron in mice and rats with Duphar B.V. (Dr. Art Tomerlin) and EPA, and reviewed notes from Oregon State University toxicologists who also discussed these tests with Duphar B.V. (Oregon State University 1985). The mouse study was conducted for Duphar B.V. by an independent laboratory in Huntingdon, England; the rat study was conducted by Hazelton Laboratories in Virginia. Both studies involved treating the test animals with 0, 16, 80, 400, 2,000, 10,000 ppm technical grade diflubenzuron in their diet. The mouse study ran for 91 weeks while the rat study ran 104 weeks. Neither study produced any evidence of carcinogenic effect for any tested dose. For example, the mouse study showed that the highest dose (10,000 ppm) produced the same incidence of tumors as were found in the untreated control. Based on proposed guidelines for Carcinogen Risk Assessment recently promulgated by EPA (USEPA 19841), diflubenzuron would be classified as "no evidence of carcinogenicity for humans" because there was no evidence for carcinogenicity in at least two adequate animal tests in different species.

However, some concern about carcinogenicity may still be expressed because 4-chloroaniline, a metabolic breakdown product of diflubenzuron, has been claimed to be a carcinogen (see Appendix G, comment letters 12u and 13kk). EPA (1979) reported that 4-chloroaniline was mutagenic in Ames tests. NCI has conducted feeding tests with rats and mice (USEPA 1979). They concluded that sufficient evidence was not found to establish the carcinogenicity of 4-chloroaniline for rats or mice. However, since some rare tumors were noted in treated animals (not significant compared to control), some uncertainty still exists about the carcinogenicity of 4-chloroaniline. Because of this uncertainty, cancer risk associated with 4-chloroaniline is discussed further in the Evaluation of Risk section.

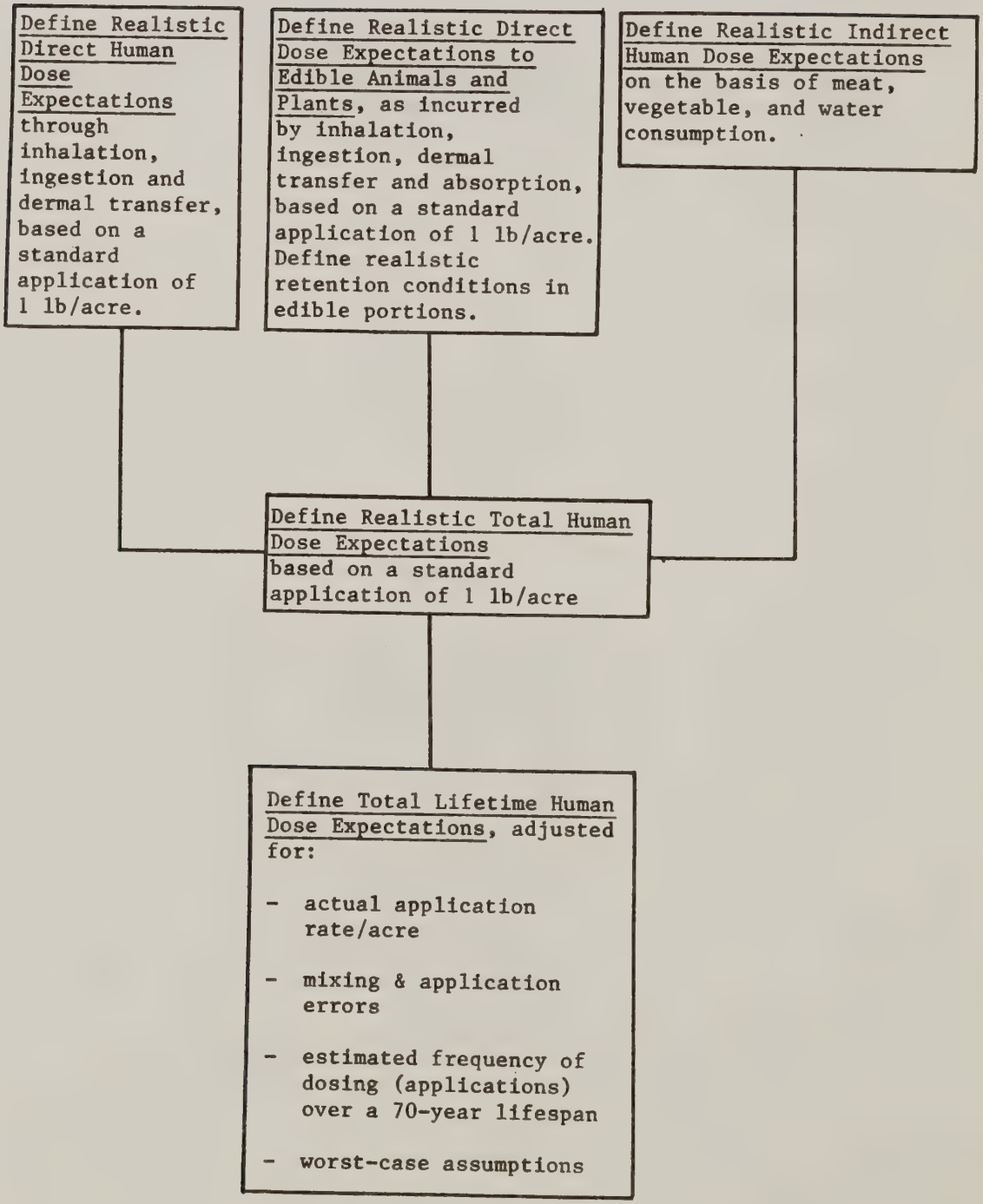
Other agencies, such as EPA, use more complicated mathematical extrapolation models which relate carcinogen exposure to cancer risk at the extremely low doses normally encountered in the environment. For example, EPA uses a multistage mathematical model to describe the linear nonthreshold dose-response relationship at low doses (USEPA 1980b). This

model incorporates a procedure for estimating the largest possible linear slope (potency) at the 95 percent confidence limit. Various models could be used which would produce different cancer risks. We chose the simplified linear model because linear non-threshold models are easier for the decisionmaker or other readers to understand. Also, risk assessments based on these models are regarded as conservative, that is estimating the upper limit for the risk (i.e., the true risk is not likely to be higher than the estimate, but it could be smaller).

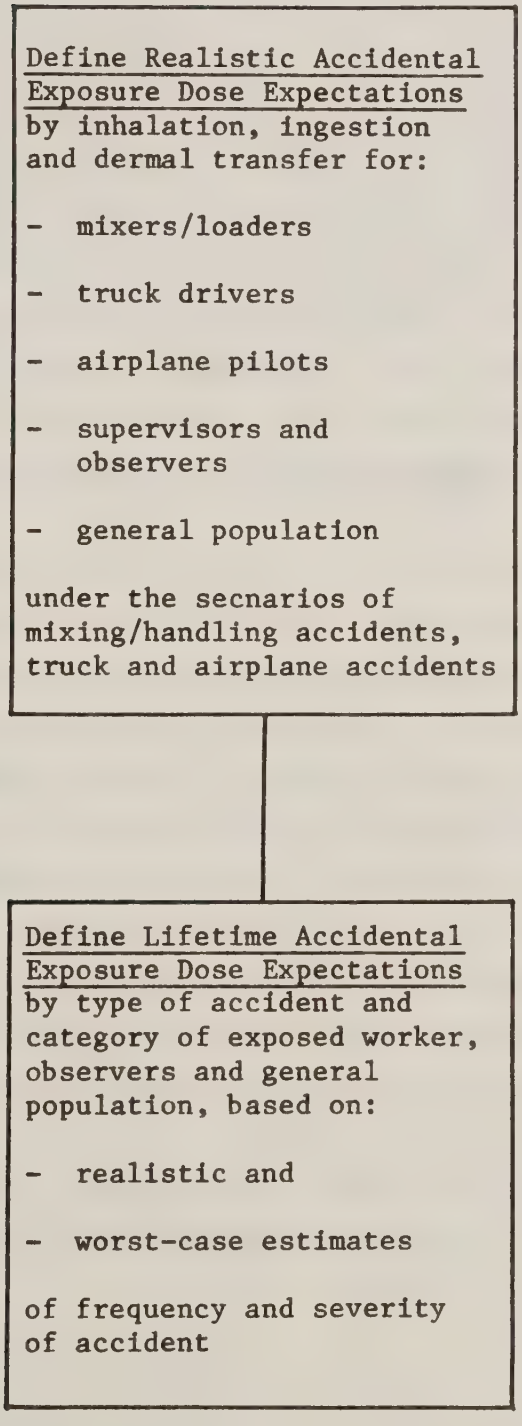
EXPOSURE ESTIMATES
Workers and Observers



EXPOSURE ESTIMATES
General Population



EXPOSURE ESTIMATES
Accidents



ANALYSIS OF EXPOSURE

General Assumptions

In this analysis, the exposure levels were first estimated for a "generic" single application of 1 lb a.i./acre applied in 1 gallon of total formulation. Exposure levels for specific insecticide applications can be calculated by multiplying the application rate (e.g., 0.75 lb a.i./acre for acephate) of the insecticide times the generic exposure level. The assumption behind this calculation is that insecticide deposits are dependent upon the volume applied, concentration, and application method. The application methods for the different insecticides are basically the same for the gypsy moth control programs. Applications are generally made with small to moderate sized fixed-wing aircraft or helicopters. The insecticides are applied using a fine, 100 to 150 micrometer (um), droplet size under meteorologic conditions that will minimize drift.

The organisms considered in this risk analysis include humans, goats, rabbits, and fish. The latter three represent sources of human food and serve as models for insecticide exposures from eating game and domestic animals. The goat, since it has a high surface area to body weight ratio, is used to represent larger animals. Rabbits which also have a high surface area to body weight ratio were chosen to represent small game animals. Rabbits were also used to determine adverse effects to domestic pets such as dogs and cats. It is important to note that rabbits and goats were used only as surrogates for determining levels of exposure and not as models for calculating toxicological effect. The surface to body weight ratio is used to determine the level of dermal exposure, the major route of exposure for game or domestic animals. Fish were included because of the potential for insecticides to be unintentionally applied or spilled into water. Estimated exposure values are expressed in terms of dose (mg/kg of body weight) entering the organism each day.

Humans and wildlife may be exposed to insecticides by skin contact (dermal route), breathing (inhalation), or eating and drinking (ingestion). These three routes of exposure include virtually all exposure situations. Fish are exposed by total immersion, a combination of all exposure routes.

Exposures to humans can be environmental or occupational. Environmental exposures refer to those incurred directly by (1) persons residing in the area during application, or (2) walking through treatment areas after application. Environmental exposures also occur indirectly by consuming animals and/or vegetation taken from a treated area. Occupational exposures refer to those incurred by three groups of workers associated with insecticide application: pilots, mixers/loaders, and ground observers. Exposure data from actual field tests were used to determine doses that could realistically occur during insecticide application. To get a range of exposure values up to and including the extreme, or worst case, some assumptions were made. In each case, the specific assumptions are spelled out. The following exposure levels are compared to toxicological data and fully discussed in the section on Evaluation of Risk.

Human Exposures and Estimated Dose Levels

Workers

Carbaryl is the only one of the four insecticides for which data was found for occupational exposures or doses incurred by workers on spray projects. The South Carolina Epidemiologic Studies Center (SCESC 1978 and 1979) determined the doses of carbaryl incurred by various occupational groups and residents following application to control spruce budworm. The New Jersey Department of Health, Epidemiology Studies Program (Schulze et al. 1979) did a similar study for the gypsy moth. In both studies, the dose values were based on urinary analysis for 1-naphthol, a breakdown product of carbaryl, and resulted from all exposure routes.

Schulze et al. (1979) reported an average 312 ppb 1-naphthol in the urinalyses of pilots and mixer/loader groups with a high of 1,268 ppb.

Data for the SCESC (1978 and 1979) are summarized in Tables 3 and 4 in the FEIS. The 1-naphthol excreted in urine can be converted to carbaryl dose by multiplying the 1-naphthol residue level by the ratio of molecular weight (201 mol. wt. carbaryl/144 mol wt 1-naphthol = 1.39) by 3, and by the average urine excreted. The multiplier 3 was used because the Union Carbide Corporation found that 32.8 percent (1/3) of the cumulative carbaryl dose is excreted as 1-naphthol during the first 12 hours (the sampling period in the tests) (SCESC 1978). Therefore, the high range of 1-naphthol, 1,268 ppb, reported by Schulze et al. (1979), translates to 2.940 mg of carbaryl (1.268 mg/l x 1.39 x 3 x 0.556 liters urine) or a dose to a 70 kg worker of 0.042 mg/kg (2.940 mg/70 kg). Similarly, the average exposure to loaders reported by SCESC (1978) (FEIS 1984, Table 3) translates to 0.029 mg/kg (1.144 mg/l x 1.39 x 3 x 0.422 l x 1/70 kg). The value of 0.042 mg/kg/day is used in this analysis as the realistic exposure value for the mixer/loader group. Lavy and Mattice (1984), Lavy et al. (1982), and Leng et al. (1982) found exposures to mixer/loaders for 2,4,5-T, 2,4-D, paraquat, MSMA (monosodium methanearsonate), and EPN (o-ethyl-o-(4-nitrophenyl) phenylphosphonothioate) could range from 0.012 to 0.127 mg/kg. Based on these 6 studies for various pesticides, it was assumed that in the worst case, doses to mixer/loaders could range up to 0.1 mg/kg/day for any of the insecticides. Exposures to pilots were roughly one-third the average exposure received by mixer/loaders or an average of 0.012 mg/kg (0.324 mg 1-naphthol/liter urine x 1.39 x 3 x 0.609 liter urine x 1/70 kg person). All occupational exposure to project personnel other than observers was assumed to be equal to that received by the highest exposure group (mixer/loaders).

Observers

The values reported by Schulze et al. (1979) and SCESC (1979) for project observers such as inspectors, scouts, rangers, and ecologists provide a realistic estimate of exposure to project observers or residents who are outside during the spray operation. Average 1-naphthol residues found in

urine was 13.48 to 51.87 ppb with the highest individual value being 247 ppb. These 1-naphthol values translate to carbaryl dose levels of 0.0004 and 0.0019 mg/kg/day (0.01348 mg 1-naphthol/liter urine x 1.39 x 3 x 0.563 liter urine x 1/70 kg person and 0.05187 mg/liter x 1.39 x 3 x 0.611 liter x 1/70 kg). The high value of 0.0019 mg/kg/day is rounded up to 0.002 mg/kg/day and this figure is used as the realistic exposure value for the observer group.

A more extreme exposure can be calculated assuming that a 70 kg person receives a direct application and that the area of exposed skin is 2 ft^2. For the "generic" application rate of 1 lb a.i./acre, approximately 10 mg will be deposited on each ft^2 of exposed skin (1.0 lb/acre x 454 g/lb x 1,000 mg/g x 1 acre/43,560 ft^2). To convert dermal exposure to dose, the dermal absorption rate of each insecticide must be applied to the exposure value. In the absence of laboratory data, the dermal absorption rate can be estimated by dividing the oral LD_{50} for mammals by the dermal LD_{50} for mammals (Eto 1977). Using this method, the dermal absorption rates are estimated to be 8.4 percent for acephate (866 mg/kg ÷ 10,250 mg/kg, Meister 1983), 5.2 percent for carbaryl (500 mg/kg ÷ 9,500 mg/kg, Union Carbide 1978) and 7.2 percent for trichlorfon (144 mg/kg ÷ 20,000 mg/kg, Mobay Chemical Corporation 1981). The dermal toxicity of diflubenzuron is of such a low order that the specific LD_{50} value is not available. For the purpose of this risk analysis, a 10-percent dermal absorption value is used for all insecticides. Therefore, the observer will receive a worst case dose of 0.029 mg/kg/day (10 mg/ft^2 x 0.1 x 2 ft^2/person x person/70 kg) for each pound of insecticide applied per acre using a 10 percent dermal absorption factor.

It is noteworthy that Feldman and Maibach (1974) reported a 73.9 percent dermal absorption rate of carbaryl when the insecticide was administered in an acetone solution. Dermal absorption rates can vary depending upon solvents used, duration of exposure, and the capability of these solvents to penetrate the skin directly, or to alter the physical condition of the skin, and thereby facilitate penetration. The use of a 10-percent dermal

absorption rate in this risk analysis is based upon the results of carbaryl exposure studies conducted by Schulze et al. (1979) and SCESC (1978, 1979). If a 73.9 percent dermal absorption rate was used to calculate dosages, observers would receive 0.22 mg/kg (10.4 mg/ft^2 x 2 ft^2 x 0.739 ÷ 70 kg). This value is more than 5 times the highest dose actually recorded in mixer/loaders (the highest exposed project worker group, under actual carbaryl application conditions in gypsy moth projects). Therefore, the use of a 73.9-percent dermal absorption rate would grossly overestimate the dermal dose observed from actual field data. Furthermore, acetone is not used as a carrier in formulations used for gypsy moth control.

General Public

Exposure studies by Schulze et al. (1979 and undated), SCESC (1978 and 1979), Maitlen et al. (1982) were reviewed to estimate exposure to residents who live in treatment areas. Schulze et al. (1979) reported finding no detectable exposure to residents in the spray area. Schulze et al. (undated) reported exposure values ranging from nondetectable to 35 ppb. The resident exposure values found by SCESC (1978 and 1979) range from nondetectable to 2,556 ppb, with only 20 percent of the residents indicating measurable amounts. The one high value of 2,556 ppb was for a resident who reported using carbaryl on his own garden. Since this individual admitted using carbaryl around the home, the 2,556 ppb value was not used to analyze exposures to residents that result from gypsy moth projects. However, this value is used later in this analysis to evaluate the effects of accumulated exposures from various sources. The next highest exposure level reported for residents was 247 ppb which translates to 0.005 mg/kg (0.247 mg/liter x 1.39 x 3 x 0.338 liters urine x 1/70 kg). The average exposure level reported for residents excluding the 2556 ppb individual was 131 ppb which translates to 0.002 mg/kg (0.131 mg/liter x 1.39 x 3 x 0.297 liters x 1/70 kg). These values include dermal and inhalation exposure as well as secondary exposures from insecticide residue on grass, foliage, cars, yard items, etc., because it is based upon the recovery of insecticide residue in urine. Urine residue levels represent

total exposure by all routes of exposure over periods of up to 36 hours following application. Maitlen et al. (1982) reported 0.3 mg/hr exposure to a bystander from applications of carbaryl to orchards. Assuming 8 hours exposure and a 10-percent absorption rate, a 70 kg person would be exposed to 0.0034 mg/kg (0.3 mg/hr x 8 hr x 0.1 x 1/70 kg). Based on these data, it was assumed that doses to residents could range from 0.002 (realistic) up to 0.005 mg/kg/day (worst case). However, exposures could be as high as those for a project observer (0.029 mg/kg/day worst case) if a resident was outdoors and received a direct application.

The SCESC data (1978 and 1979) (FEIS, Tables 3 and 4) show that exposure of project scouts (observers) who were not exposed to a direct application but who entered the spray areas immediately after application ranged from 0.0004 to 0.0009 mg/kg. Therefore, it is assumed that exposure of individuals who are indoors during application, but who receive indirect exposure to insecticide residues, is below 0.0009 mg/kg.

Exposure of residents who live adjacent to or near treatment areas can result from drift. Witt (1984) reviewed a number of drift studies (which referred to 75-100 um drop size) and reported drift values of 2, 0.5, and 0.15 percent of the amount deposited on targets at distances from the application site of 1/4, 1/2, and 1 mile, respectively. These values are used for calculating doses received from insecticidal drift because they are based on actual drift data, and involve droplet sizes typical of gypsy moth spray programs. Extrapolation of this data for near range drift (250 feet from the treatment area) shows the dose to be approximately 67 percent of that for a resident in the treatment area. For this analysis then, the realistic and worst case doses for near range drift is calculated to be 0.0013 mg/kg (0.002 x 0.67), and 0.0033 mg/kg (0.005 x 0.67), respectively.

Exposure by inhalation has already been estimated in the occupational and residential exposure values. They are included in the results of urine analysis which represents exposure by all routes.

In summary, the estimated doses used in this analysis are:

	Realistic	Worst Case
Workers		
- Mixers/loaders	0.042 mg/kg/day	0.1 mg/kg/day
- Observers	0.002 mg/kg/day	0.029 mg/kg/day
Residents		
- Direct exposure	0.002 mg/kg/day	0.005 mg/kg/day
- Near range drift	0.0013 mg/kg/day	0.0033 mg/kg/day
- Indirect exposure	0.0004 mg/kg/day	0.0009 mg/kg/day

Animal Exposures and Estimated Dose Levels

Exposures to animals can be estimated by the following method using specific assumptions.

Dermal

Dermal exposure to insecticides for animals (wildlife, livestock, domestic) in the treatment areas is a function of the deposition rate on the animal and of the surface area contacted by the deposited spray. Smaller animals represent a proportionately larger surface area per unit weight and thus should have higher exposure levels.

Animals are found in either rural or suburban areas where gypsy moth suppression or eradication projects are conducted. Ground level deposition from aerial application of insecticides is partially shielded by overstory foliage and is approximately one-tenth to one-third of that at the overstory (Maksymiuk and Orchard 1975, Newton and Dost 1981), or 10 to 35 mg/m^2 for a 1 lb a.i./acre application at ground level where it would be deposited on animals.

Goat dermal exposure and dose assume the following:

- the goat weighs 40 kg (88 lbs.) and has 2.3 m^2 surface area (USDA 1984b).

- the dorsal surface is contacted by deposited spray.

- treated foliage contacts the goat's sides and abdomen as the animal walks through it, at a concentration equivalent to direct deposition. It is assumed that 100 percent of the residue on foliage can be dislodged.

- dermal absorption rate ranges from 1 to 10 percent. A 10 percent absorption rate is considered to be worst case assumption because dermal toxicity studies are conducted on shaved animals. Animal fur acts like human clothing protecting against dermal exposure.

The estimated dermal exposure to a goat is 2.02 mg/kg (35 mg/m^2 x 2.3 m^2/goat x goat/40 kg) for each 1 lb a.i. applied per acre. The estimated dermal dose to the goat ranges from 0.020 to 0.202 mg/kg (2.02 mg/kg x 0.01 or 0.1) depending on the dermal absorption rate.

Rabbit dermal exposure and dose assumes:

- the rabbit weighs 2.0 kg and has 0.23 m^2 surface area (USDA 1984b).

- the dorsal surface is contacted by deposited spray.

- the ventral surface is contacted by treated foliage as the animal walks through it, with an insecticide concentration equivalent to direct deposition.

- dermal absorption rate ranges from 1 to 10 percent, with the latter rate considered a worst case assumption for reasons previously stated. The estimated dermal exposure to rabbits is 4.03 mg/kg (35 mg/m^2 x 0.23 m^2/rabbit x rabbit/2 kg) for each 1 lb a.i. applied per acre. The estimated dermal dose to rabbits ranges from 0.040 to 0.403 mg/kg (4.03 mg/kg x 0.01 or 0.1).

Inhalation

Inhalation exposures of animals in treatment areas would occur on a one-time basis and would be limited to a short time (measured in minutes at least). Lavy and Mattice (1984) showed that dermal exposure accounts for 99.8 percent of the exposure in humans. Exposure by inhalation is therefore 0.2 percent. Assuming that inhalation by animals is similar to humans, exposure by this route in animals would therefore be expected to be so small that it would be within the error of the dermal exposure calculations. It is subsequently excluded from analysis.

Immersion

Exposure to fish is based on two major assumptions: 1) direct applications could inadvertently be made to streams or ponds during insecticide application, and 2) the minimum average water depth would be 6 inches. An unintentional direct application to open water will result in 10 mg/ft^2 for a 1 lb a.i./acre application (454 g/lb x 1000 mg/g x 1 lb/acre x 1 acre/43,560 ft^2). For a water body with a depth of 6 inches, the resulting concentration would be 20 mg/ft^3 or 707 mg/m^3 assuming no dilution of water from sources outside the treatment area. This is equivalent to a concentration of 707 ppb (707 mg/m^3 x m^3 water/10^9 mg) in water for every pound of active ingredient applied per acre. In comparison, as stated in the FEIS (p. 45-47), actual residue values resulting from 1 lb a.i./acre applications of carbaryl range from 30 to 80 ppb. Exposure and subsequent dose to fish are therefore assumed to range from a realistic value of 50 ppb to a worst case value of 707 ppb for every 1 lb a.i./acre applied.

It should be emphasized that the calculated doses to fish are deliberate overestimates, since they are based upon the assumption that the water receives a direct application of insecticide. This act in itself can be considered a worst case occurrence since direct application is avoided during gypsy moth control projects. The calculated worst case dose (707 ppb) is more than 10 times the realistic dose actually measured in water that was intentially treated with insecticide. The realistic dose by itself could be considered the worst case dose.

Oral

Animals may be exposed to insecticides as a result of ingesting plant material, water, and during the course of grooming. Since insecticides are usually rapidly degraded (see Table 2, FEIS) or, if not degraded, translocated to often inedible plant parts, exposure to animals by ingestion of plants will be only a short-lived phenomenon. Studies of residues on vegetable crops or grass illustrate that initial residues of insecticides range from 1 to 100 ppm depending on the insecticide and type of the vegetation (see, for example, Pieper 1979, US EPA 1983a, Back 1961, and Kuhr and Dorough 1976). These residues degrade to nondetectable levels within 10 to 14 days on vegetation except for grass which can have detectable residues for up to 28 days (Pieper 1979).

In some cases all exposures were not considered because the contribution to the total dose value is insignificant. For example, exposure via inhalation (see p. F-34) contributes only 0.2 percent to the total dose. It should be noted that animals may consume some amounts of insecticide if they lick dewfall from foliage that has recently been sprayed; however, such ingestion is difficult to quantify. In addition, oral exposure during the course of grooming is difficult to quantify, although it is more likely to occur in small grooming mammals and preening birds than in goats. The areas that these mammals tend to groom, such as the belly, are also less likely to have higher insecticide concentrations than the back.

Goat and rabbit oral exposure and dose assume:

- post-spray concentration of insecticides in browse and food foliage ranges from 10 ppm (realistic) to 100 ppm (worst case).

- a typical goat weighs 40 kg (88 lbs) and eats 1.15 kg (2.5 lbs) field weight foliage per day. A typical rabbit weighs 2.0 kg, eats 60 g of food (dry weight) daily, and receives a dose of 0.03 mg/kg/day for each ppm in food (USDA 1984b).

- oral dose equals oral exposure.

Thus, the realistic and worst case exposures for a goat due to ingestion would be 0.29 mg/kg/day (10 mg/kg x 1.15 kg/goat/day x goat/40 kg) and 2.88 mg/kg/day (100 mg/kg x 1.15 kg/goat/day x goat/40 kg) for each 1 lb a.i./acre applied. Estimated doses are the same (0.29 and 2.88 mg/kg/day, respectively).

Similarly, a typical rabbit would have realistic and worst case exposures of 0.3 mg/kg/day (10 ppm x 0.03 mg/kg/day/ppm) and 3.0 mg/kg/day (100 ppm x 0.03 mg/kg/day/ppm) for each 1 lb a.i./acre applied. Estimated doses are the same (0.03 and 3.0 mg/kg/day), respectively.

In summary the estimated doses for animals are:

		Realistic	Worst Case
Dermal			
Coats	-	0.02 mg/kg/day	0.20 mg/kg/day
Rabbits	-	0.04 mg/kg/day	0.40 mg/kg/day
Oral			
Goats	-	0.29 mg/kg/day	2.88 mg/kg/day
Rabbits	-	0.30 mg/kg/day	3.00 mg/kg/day
Immersion			
Fish	-	0.05 mg/kg/day	0.71 mg/kg/day

Environmental Exposure to Humans and Estimated Dose Levels

Individuals in treatment blocks, or persons downwind of the application site, may be exposed to insecticides by dermal contact, inhalation, or ingestion. However, as seen by the data presented previously, dermal and inhalation exposures to persons downwind are low relative to those experienced by project personnel. For example, residents or casual visitors to spray blocks would not be expected to receive a dermal exposure higher than project personnel who are in the treatment area and receive direct spray deposition. Consequently, estimates of occupational and resident exposures to the insecticides, which have already been presented and discussed, include both dermal and inhalation exposure because they are based upon insecticide monitoring studies. Therefore, these exposure estimates are sufficient for both realistic and worst case exposures. Environmental dose levels to humans by dermal and inhalation routes were not considered further. However, since any human may eat vegetation (fruits or vegetables) and meat (domestic or game) that may contain insecticide residues, environmental exposure levels by the oral route are considered further.

Oral exposures to humans occur through ingestion of fish, meat, vegetation, and water. The levels of exposure are a function of insecticide concentration in the food or water as well as the quantity of material consumed.

There are no data available documenting the amounts of carbaryl, trichlorfon, diflubenzuron, or acephate in food animals that are in areas treated for gypsy moth. There are, however, a number of studies documenting pesticide levels in animal tissue, milk, or eggs resulting from direct application to animals for insect control purposes. For example, carbon 14 (C^{14}) labeled carbaryl fed to cattle in doses of 0.25 and 3.05 mg/kg resulted in radiolabeled residues in milk equivalent to 0.35 percent of each dose (Dorough 1967). Maximum concentrations of carbaryl-C^{14} equivalents (a total of all metabolites) of 0.06 ppm and 0.95 ppm were

found in milk 6 hours after administering the 0.25 and 3.05 mg/kg doses respectively. Analysis of 27 tissues (muscle, organs, or parts thereof) sampled 6 days after the high dose showed only trace amounts of residues (about .0003 mg/kg based on the radioactive counts).

In a similar study involving diflubenzuron (Nye 1976), lactating dairy cattle were given daily dose equivalents ranging from 0.05 to 250 ppm. The total radiocarbon residues for the 250 ppm dose were less than detectable limits (0.34 ppm) while the kidney and liver (organs that remove foreign chemicals from the body) contained 1.06 and 6.04 ppm respectively after 8 days of exposure. Other animal residue studies demonstrate that trichlorfon (no metabolites were analyzed for) was nondetectable (less than 0.05 ppm) in fat, heart, kidney, liver, and muscle of cows fed up to 325 ppm trichlorfon for 28 days (USEPA 1983a). Pour-on applications of trichlorfon to cattle also resulted in nondetectable residues (USEPA 1983a).

Chevron Chemical Co. (Chevron Chemical Co. 1973) reported that most acephate and its principle metabolite O, S-dimethyl phosphoramidothioate (Monitor^{R}) was excreted from rats and goats within 12 hours. Only trace levels of the C^{14} labeled acephate were detected in tissues during dosing. These levels declined rapidly to zero when dosing ceased. Also, applications of 0.7 lb a.i. acephate/acre in New York (LOTEL 1975) resulted in non-detectable levels (less than 0.05 ppm) of acephate in the liver of muscle tissue of rodents trapped in the treated area. Such studies suggest that residue levels in goats, rabbits, or other animals that might be in an area treated for gypsy moth would most likely be below detection limits. These levels would certainly never exceed residue levels detected in animals that were deliberately fed insecticides or to which insecticides were topically applied.

For this analysis then, it was estimated that residue levels in animals could range from 0.0003 mg/kg of tissue to about 0.3 mg/kg. Except for diflubenzuron, which is somewhat fat soluble, any residues that might be in muscle tissue would be eliminated by the animal within a few days (USEPA 1983a and Kuhr and Dorough 1976). For this analysis, a 1-week elimination period was assumed.

Consumption of Fish

Oral dose estimates for human consumption of fish, which are exposed through the single route of immersion, assume:

- insecticide concentrations in water range from a realistic value of 50 ppb to a worst case concentration of 707 ppb per 1 lb a.i./acre applied under normal operating conditions.

- the low octanol/water partition coefficients, 0.04 for acephate, 3 for trichlorfon, and 240 for carbaryl, indicate that these insecticides should not be fat soluble or accumulate in fish tissue. The relatively high octanol/water partition coefficient for diflubenzuron (5,000) indicates that it could bioaccumulate, although studies by Schooley and Quistad (1979) indicate that bioaccumulation should be minimal. Diflubenzuron levels in fish tissue rapidly drop toward zero once the fish are placed in fresh water. Nevertheless, a bioconcentration factor of 1.0 for diflubenzuron is assumed in this analysis. Studies reported in Kuhr and Dorough (1976) show that the bioconcentration factor for carbaryl is less than 0.1. Since carbaryl has the highest octanol/water partition coefficient of these three insecticides, this value is used as the bioconcentration factor for acephate, carbaryl, and trichlorfon.

- insecticide residues are evenly distributed in fish tissue.

- fish with the highest tissue concentrations are most likely to be in shallow streams and ponds.

- A reasonable expectation for fish consumption is 0.5 kg/day.

Consequently, in fish tissue, insecticide concentrations are estimated to range from 0.005 to 0.71 ppm (0.05 and 0.71 ppm x bioconcentration factor) depending upon the insecticide applied, and are equivalent to 0.005 to 0.71

mg/kg for each 1 lb a.i./acre applied. Human consumption of 0.5 kg of fish would result in doses ranging from 0.00004 to 0.0051 mg/kg/day (0.005 and 0.71 mg/kg x 0.5 kg/day/person x person/70 kg) for a 70 kg person, again depending upon the insecticide applied and its bioconcentration factor.

Consumption of Meat

Oral dose estimates for human consumption of contaminated game or domestic meat (represented by the goat and rabbit surrogates) assume:

- goat and rabbit meat (or that of animals which they represent) will come from animals that may have been in the target spray zone during insecticide application and that may have eaten sprayed vegetation.

- maximum residues of insecticides in the meat are the result of both oral and dermal dose to the animal (domestic or game), which in turn are a result of the level of exposures of the animal to the insecticides, assimilation of the insecticide, and duration of exposure.

- no loss of insecticide from the sprayed animal as the result of excretion.

- where exposure is estimated through the calculation method on pages F-32 and F-34, assimilation of the insecticide into animal tissue is 10 percent of the estimated doses to the animals.

- maximum insecticide residues are equally distributed in edible meat tissue.

- estimated daily consumption of meat totals 0.5 kg per day.

Realistic insecticide residues in goat meat are assumed to be no more than the residue value of 0.0003 mg/kg of tissue reported in the cow fed 3.05 mg carbaryl/kg body weight (Dorough 1967). The worst case residue, based on the combination of the calculated worst case dermal and oral doses assuming a 10 percent assimilation of the dose and even distribution through all tissues, is estimated to be 0.31 mg/kg [(0.202 mg/kg + 2.88 mg/kg) x 0.1] for every 1 lb a.i./acre applied. Since it is assumed that a person would eat as much as 0.5 kg of meat per day, the human dose resulting from eating contaminated meat range from a realistic value of 0.000002 mg/kg (0.0003 mg/kg meat x 0.5 kg meat eaten x 1/70 kg person) to a worst case of 0.0022 mg/kg/day (0.31 mg/kg/day x 0.5 kg/day/person x person/70 kg).

Realistic residues in rabbit meat are estimated from the cow data by considering the reduced food consumption of the rabbit compared to the goat. In other words, residues in rabbits would be 0.05 (60 grams for rabbits/1,150 grams for goat) times the residue in cows, or 0.000015 mg/kg tissue (0.0003 mg/kg x 0.05). Worst case residue levels are based on the worst case dermal and oral doses and are estimated to be 0.34 mg/kg [(0.403 mg/kg + 3.00 mg/kg) x 0.1] for every 1 lb a.i./acre applied. Since it is assumed that a person would eat as much as 0.5 kg of rabbit meat per day, the human dose resulting from eating rabbit meat containing insecticide residues would range from a realistic dose of 0.0000001 mg/kg (0.000015 mg/kg rabbit x 0.5 kg meat x 1/70 kg person) to a worst case dose of 0.0024 mg/kg/day (0.34 mg/kg/day x 0.5 kg/person x person/70 kg).

Consumption of Water

The estimated oral doses that result from a person consuming water that contains insecticide residues assume the following:

- direct application of insecticide to water (contrary to normal operating procedures).

- water sources will have a minimum average depth of 6 inches.

- realistic insecticide concentrations are 50 ppb (0.05 mg/liter) for every 1 lb a.i. per acre application. Worst case concentrations are 0.707 mg/liter) for every 1 pound a.i. per acre application of insecticide.

- daily consumption of water is 2 liters.

- water consumed is from a surface spring, pond, or stream that had direct application.

- residue persists for a maximum of 5 days (see FEIS p. 65).

- it is possible that after spray application, some insecticide might be dislodged by rain within 10 days (based on half-life data) (FEIS, Table 2), and runoff into potable water. This may result in a brief increase in the concentration of insecticide in water. The transitory nature of these residues and the relatively small contribution of drinking water to human exposure compared to the dermal exposure values already estimated (p. F-32) indicate that runnoff is not a significant contributing factor for exposure and is thus not considered in this analysis.

Thus, the human exposure of insecticides contributed by drinking water would range from a realistic value of 0.0014 mg/kg/day (0.05 mg/kg x 2 liters/day x person/70 kg/person) to worst case dose of 0.020 mg/kg/day (0.707 mg/liter x 2 liter/day x person/70 kg).

Consumption of Fruit and Vegetables

Ingestion of vegetables or fruits that contain insecticide residues must be considered negligible. During the time of the year when gypsy moth control is initiated, no fruit and only limited greens would be available. Weather conditions that speed up plant development also would mean earlier insecticide applications and visa versa. Since no data were available for

insecticide residues on fruits or vegetables resulting from the use of insecticides to control gypsy moth, residue data from agricultural applications was used as a surrogate.

Kuhr and Dorough (1976) report on a number of studies involving the biostability of carbaryl on crops. The spinach group of crops had initial residues of 52 ppm that degraded to about 9 ppm in 1 week. Lettuce residues were usually lower and dissipated shortly after application partly because of dilution by growth. Residues of trichlorfon were reported (USEPA 1983a) to be nondetectable 14 days after treatment with 1.72-2.00 lb a.i.,/acre. Chevron Chemical Co. (1973) reported residues of acephate ranged from 4.3 to 12.4 ppm on lettuce and broccoli measured 3 days after 2 lb/a.i./acre treatments. These residues degraded to 1.96 to 4.11 ppm 14 days after treatment. Such data indicate that initial insecticide residues could range from 10 to 50 ppm, but degrade rapidly to nondetectable values within 1 or 2 weeks. Kuhr and Dorough (1976) also reported that simple washing shortly after spraying removed more than 90 percent of carbaryl residues.

Therefore, vegetables were assumed to have an initial range of residues from 1 to 5 ppm after washing, and these residues degrade to zero (undetectable) within 14 days. Since acephate has a unique characteristic (of the four insecticides being analyzed) of being adsorbed into or onto leaf surfaces, only 5 percent acephate residues can be removed by washing with water if the residues have been on longer than one day. One hour after treatment 30 percent of the residues can be removed by wiping (and probably washing). This analysis assumed that only 5 percent of acephate residues would be removed through washing. Therefore, acephate residue levels would range from 4.0 to 11.4 ppm after washing. Assuming a person eats 0.5 kg of vegetables per day, the human dose contributed by contaminated vegetables would range from 0.007 mg/kg (1 mg/kg residue x 0.5 kg x 1/70 kg person) to 0.035 mg/kg (5 mg/kg x 0.5 kg x 1/70 kg), if the vegetables were picked and eaten the same day as the application (0.028 to 0.081 mg/kg if the vegetables contain acephate residues). If the vegetables were picked on subsequent days, doses would be considerably less because of pesticide degradation.

Summary of Expected Doses

Calculated dose levels for humans are based upon an application rate of 1 lb a.i./acre applied and are summarized below. Dose levels for both realistic and worst case were calculated using the highest estimated exposure values. Therefore, these levels represent maximum exposure doses and are not an average. An increase or decrease in the application rate is assumed to produce a proportionate change in the calculated dose level.

Dermal and Inhalation Doses for Humans

	Realistic	Worst Case
Workers		
- Mixers/loaders	0.042 mg/kg/day	0.1 mg/kg/day
- Observers	0.002 mg/kg/day	0.029 mg/kg/day
Residents		
- Direct exposure	0.002 mg/kg/day	0.005 mg/kg/day
- Near range drift	0.0013 mg/kg/day	0.0033 mg/kg/day
- Indirect exposure	0.0004 mg/kg/day	0.0009 mg/kg/day

Oral Doses for Humans

	Realistic	Worst Case
- Eating fish 1/	0.0004 mg/kg/day	0.0051 mg/kg/day
- Eating rabbit	0.0000001 mg/kg/day	0.0024 mg/kg/day
- Eating goat	0.000002 mg/kg/day	0.0022 mg/kg/day
- Drinking water	0.0014 mg/kg/day	0.020 mg/kg/day
- Eating vegetables and fruits	0.007 mg/kg/day	0.035 mg/kg/day

1/ Realistic and worst case doses given are for diflubenzuron and represent a bioconcentration of 1.0 in fish. The realistic and worst case doses for acephate, carbaryl, and trichlorfon are calculated by multiplying the doses identified for diflubenzuron by 0.1, the estimated bioconcentration factor for these products in fish (see p. F-39 amd F-40 for rationale). Hence, the realistic dose becomes 0.0004 x 0.1 or 0.00004 mg/kg/day, and the worst case dose 0.0051 x 0.1 or 0.00051 mg/kg/day.

For each pound active ingredient of insecticide applied, the realistic dose received from a daily diet consisting of 0.5 kg of meat (or fish), 0.5 kg of vegetables and fruits, and 2 liters of water is estimated to be 0.0089 mg/kg/day (0.0004 + 0.0071 + 0.0014) for diflubenzuron, 0.0085 mg/kg/day (0.00004 + 0.0071 + 0.0014) for carbaryl, and trichlorfon and 0.029 mg/kg/day (0.00004 + 0.028 + 0.0014) for acephate.

Similarly, the worst case dose received from such a diet is estimated to be 0.061 mg/kg/day (0.0051 + 0.0357 + 0.0202) for diflubenzuron and 0.058 mg/kg/day (0.0024 + 0.0357 + 0.0202) for acephate, carbaryl, and trichlorfon, and 0.103 mg/kg/day (0.0024 + 0.081 + 0.020) for acephate.

Variations to the Applied Dose

The amount of insecticide actually applied to an area is subject to normal and abnormal variations that may occur during mixing and application. The normal variations account for minor errors (+ or -) in the actual dose rates applied and are common to all pesticide applications whether they be aerial or ground applications, or application of pesticides around the home. These normal variations are used as a conservative estimate in that the highest level of the error range (+) is assumed to occur under all conditions. For the purpose of this analysis, only factors that account for dose increases due to normal variations were used.

The major sources of normal mixing and application variations are:

1. Quality control in insecticide manufacture.
2. Errors of measurement during mixing.
3. Excessive swath overlap during application.
4. Improper aircraft calibration.
5. Drift off target.

The actual amount of active ingredient in a chemical insecticide formulation may vary from the label by ±4 percent according to EPA regulations. Consequently, in actual use situations, it is conservatively

estimated that up to 4 percent more insecticide could unknowingly be applied. Other normal variations take place during the field mixing phase and result from minor calibration variations in flow meters used to measure amounts of insecticide or the diluting agents.

Excessive spray swath overlap, skips between swaths, and drift off target may occur, but all except the first one will result in less insecticide actually being deposited in the target area. The assumption in this analysis that the insecticide is completely and evenly distributed over the target area takes into account these minor variations. Improper aircraft calibration can result in either more or less insecticide than planned being applied over the target area. Standard procedures implemented on projects call for recalibration of aircraft if application rates exceed ±5 percent of calibrated rates. Consequently, up to 5 percent more material could be applied to an area under normal operational conditions. A conservative estimate for the extremes of the error range attributable to all normal operational variations is therefore assumed to be ±10 percent. Extremes in quality control measurement and aircraft calibration errors would all have to occur simultaneously to reach this level, rather improbable, but possible. For this analysis, the assumption was made that up to 10 percent more insecticide than planned is actually applied. Therefore a 1.1x error factor is applied to the realistic doses listed on p. F-44 to account for normal variations.

Abnormal variations can account for major differences (+ or -) in the actual dose rates applied. For the purpose of this analysis, abnormal variations are treated as an increase in the worst case doses listed on p. F-44, but with an estimated probability of occurrence. Aircraft and truck accidents that result in insecticide spills are also treated in a similar manner, but are presented as special cases.

The major sources of abnormal operational variations are:

1. Use of an insecticide not scheduled for a particular area.
2. Treatment of an area not scheduled for treatment.
3. More than the scheduled number of applications to an area.
4. Major errors in mixing.

For use of an unscheduled insecticide, and treatment of an area not scheduled for treatment (variations #1 and #2), the worst that could happen is that a single dose of material would be applied. Therefore, the dose applied would be the estimated realistic dose (p. F-44) times the 1.1x error factor for normal variations.

For more than the scheduled number of applications to an area (variation #3), it is assumed that a double application is made to an area scheduled to receive a single application. Major mixing errors (variation #4) are also possible. The probable occurrence is one in which 2 times the amount of insecticide is mixed in the batch tank. Therefore, for variations #3 and #4 the dose applied would be the estimated worst case dose (p. F-44) times a 2x error factor.

To calculate the expected doses received from each insecticide, the realistic or worst case dose levels (p. F-44) are multiplied by the maximum registered application rate of each insecticide and either the realistic application error factor (1.1x) or the worst case error factor (2x) previously described. For example, the expected realistic dose for a member of the general public directly exposed to acephate is 0.0017 mg/kg/day [(0.002 mg/kg/day x 0.75 (application rate) x 1.1 (realistic application error factor)]. Similarly, the expected worst case exposure is 0.006 mg/kg [(0.004 mg/kg/day) x 0.75 x 2.0 (worst case application error factor)].

Probability of Abnormal Variations Occurring

The probability of abnormal operational variations occurring can be greatly minimized by implementing effective mitigation measures. Abnormal variations are directly related to mixing and application errors by project personnel, which can be minimized by implementing a well structured project. Specific measures commonly employed on gypsy moth projects include: 1) project monitors who watch the spray planes and communicate with pilots and airport supervisor, 2) loading supervisors who monitor mixing procedures and maintain loading records, and 3) project supervisors who debrief the applicators on a daily basis and orient pilots and other project personnel prior to the next day's work.

The occurrence of abnormal variations during past gypsy moth projects provides a basis for calculating the probability of such occurrences in the future. The probability of using an insecticide not scheduled for a particular area (variation #1) is nonexistent if only one insecticide is used in a project. This is generally the case in eradication projects conducted in populated areas. The greatest chance for this variation to occur is when: 1) three or more insecticides are used during a project, 2) all are being used to treat the same type of area (e.g., residential areas), 3) all are being used to treat areas in close proximity to one another, and 4) mixing and loading of all three are done at a single location by the same contractor. The gypsy moth project records for the Maryland and New Jersey Departments of Agriculture from 1982 to 1984 were used to calculate probability of occurrence since the conditions of their projects closely match those outlined above. Assuming that the average aircraft can treat about 300 acres per load, a total of 1,619 individual aircraft loads were required to treat 485,621 acres (1982 to 1984 project acreage). In neither State was there a recorded incident of using an insecticide not scheduled for a particular area (variation #1).

Incident reports from gypsy moth projects reported to the USDA Forest Service for the period 1979 to 1984 were used to estimate the probability of occurrence of treating an area not scheduled for treatment (variation #2). The record indicates that there were 3 reported cases where an area not scheduled for treatment was actually treated. During that 6 year period, more than 2.4 million acres were treated requiring more than 8,000 individual aircraft loads (assuming 300 acres treated per load). The calculated probability for the occurrence of treating an area not scheduled for treatment (variation #2) is therefore 3 in 8,000 or 1 chance in 2,667 aircraft loads. An upper limit for the accident frequency (λ_1) can be calculated by a method described by Thedeen (1979) if the accidents are assumed to occur randomly in time. If N(a) is the number of accidents for up to "a" events (trips, miles driven, etc.), the upper confidence level with a $1-\alpha$ confidence limit is calculated as follows:

$$= \frac{\chi^2_{\alpha(2(N(a)+1))}}{2a}$$

Where $\chi^2_{\alpha}(2(N[a] + 1))$ is the standard chi square distribution found in statistical tables and summarized below:

$$2\lambda_1 a$$

N(a)	$\alpha = .500$	$\alpha = 0.05$	$\alpha = 0.01$
0	1.39	5.99	9.21
1	3.36	9.49	13.28
2	5.35	12.59	16.81
3	7.34	15.51	20.10
4	9.34	18.31	23.21

For no accidents (N(a) = 0) in 1,619 aircraft loads (the historical reports for variation #1), the value of $\chi^2_{\alpha}(2(N_a + 1))$ for the 95 percent confidence limit is 5.99 and the accident frequency, λ_1, is calculated:

$$\lambda_1 = 5.99/(2 \times 1{,}619)$$
$$= 1.85 \times 10^{-3}$$

For 3 accidents (N(a) = 3) in 8,000 aircraft loads (the historical reports for variation #2), the value of $\chi^2_{\alpha}(2(N_a + 1))$ for the 95 percent confidence limit is 15.51 and the accident frequency, λ_1, is calculated:

$$\lambda_1 = 15.51/(2 \times 8{,}000)$$
$$= 9.69 \times 10^{-4}$$

To find the annual probability of accidents, the probabilities above are divided by the number of years for which the reports were made. This results in the values:

1. for variation #1

$$P = 1.85 \times 10^{-3}/2 \text{ years}$$
$$= 0.9 \times 10^{-3} \text{ or } 0.9 \text{ accident per } 1{,}000 \text{ chances per year}$$

2. for variation #2

$P = 9.69 \times 10^{-4}$/6 years

$= 1.62 \times 10^{-4}$ or 1.62 accidents per 10,000 chances per year

For variations #3 and #4, there were no historical data available from which to calculate probability. It is therefore assumed that the probability of occurrence is neither greater, nor less, than those projected for variations #1 and #2. For the purpose of this analysis, a worst case probability of 0.9×10^{-3} is assigned.

Summary of Variations to the Applied Dose

1. The dose assumptions are:

   - Realistic dose = 1.1 error factor for normal variations times the realistic doses on p. F-44.

   - Worst case dose = 2.0 error factor for abnormal variations times the worst case doses on p. F-44.

2. Probabilities of occurrence of abnormal variations at the 95 percent confidence level are:

   - Variation #1

   Use of an insecticide not scheduled for an area = 0.9×10^{-3} or 0.0009 per year (about 1 chance in 1,000)

   - Variation #2

   Treatment of an area not scheduled for treatment = 1.62×10^{-4} or 0.000162 per year (about 1 chance in 6,000)

- Variation #3

More than scheduled number of applications to an area	=	0.9×10^{-3} or 0.0009 per year (about 1 chance in 1,000)

- Variation #4

Major errors in mixing	=	0.9×10^{-3} or 0.0009 per year (about 1 chance in 1,000)

The probability of any abnormal variation occurring is the sum of these probabilities which totals 2.86×10^{-3} or 0.00286 per year. The probability for the occurrence of an abnormal variation that results in a worst case dose (2x) is the sum of the probabilities for those variations where a worst case dose is assumed (variation #3 + variation #4, see page F-47. This probability equals 1.8×10^{-3} or 1 chance in 556 per year.

Accidental Exposures

Worst possible exposures to humans, animals, or fish can result from accidental events such as large spills at the mixing/loading site or on land or into potable water sources. In this analysis, the exposure levels and subsequent doses identified are based upon worst case numbers and assumptions for aircraft and trucking accidents.

The size of the spill and the resulting impact depends on many variables such as spill source (truck or aircraft), size of insecticide load (gal.), distance to water, stream size, and density of human or animal population. Aircraft spills are assumed not to be greater than 300 gallons because that is the average load carried by the type of aircraft commonly used. Transportation spills have the potential for being much larger. A 2,000-gallon maximum was assumed because of limitations in truck capacity for the types of tank trucks used in gypsy moth suppression or eradication projects.

Aircraft-originated Spills

Other assumptions made to evaluate exposure from an aircraft spill are:

- the entire load is dumped in 15 seconds.
- aircraft speed is 100 mph.
- the spill occurs over a residential area in one case and over 2 streams in another (a 25-foot wide stream with a discharge rate of 250 cubic feet per second and a 15-foot wide stream with discharge of 70 cfs).
- the width of the spill is equivalent to the wing span of the aircraft (50 feet).

Based on the above assumptions, an aircraft spill will cover 110,000 ft^2 (100 mi/hr x 1/3,600 sec/hr x 5,280 ft/mi x 15 sec x 50 feet wide). For 1 lb. a.i./gal insecticide, the deposit on each ft^2 of surface resulting from the 300-gallon spill is 1,238.2 mg/ft^2 (300 gal x 1 lb/gal x 454 g/lb x 1,000 mg/g x 1/110,000 ft^2). If a 70 kg person were in the spill area, some physical injury could occur. The dermal exposure, again assuming 2 ft^2 exposed skin, is 35.4 mg/kg (1,238.2 mg/ft^2 x 2 ft^2/person x person/70 kg). Exposure could conceivably be greater than this because of the volume of liquid (0.03 gal/ft^2) that would also come in contact with clothing. Assuming clothing allows 25 percent of the liquid to come in contact with skin (Orlando et al. 1981, Davies et al. 1982) and the liquid is concentrated on one side of the person, the wet clothing adds 37.6 mg/kg to the exposure (1,238.2 mg/ft^2 x 0.25 x 17 ft^2 clothing/person x ½ clothing x person/70 kg). Total dermal exposure is therefore estimated to be 73.0 mg/kg (35.4 mg/kg + 37.6 mg/kg).

If the spill is directly over a stream, the mean 24-hour concentration level, or MC (24), in ppb (parts per billion) can be calculated using the following equation (USDA Forest Service 1984a):

$$MC\ (24) = 185 \times \text{lbs of insecticide/cfs}$$

To simplify calculations, it is assumed that all of the spilled insecticide would land in the stream. Actually, only half would land in a 25-foot wide stream given the 50-foot swath width of the aircraft.

For the two streams considered, MC(24) values are 222 and 793 ppb for the 25- and 15-foot streams, respectively. If a 70 kg person drank 2 liters of this water, the dose would be 0.006 mg/kg (0.222 mg/liter x 2 liters/person/day x 1 day x person/70 kg) and 0.023 mg/kg (0.793 mg/liter x 2 liters/person/day x 1 day x person/70 kg), respectively.

For aircraft spills, exposure summaries (based on 1 lb a.i./gallon mixture) are:

Dermal (partial)	-	35.4 mg/kg.
Dermal (full)	-	73.0 mg/kg.
Drinking water	-	0.023 mg/kg.

Vehicle-originated Spills

Direct exposure to project personnel from a spill involving transportation was evaluated. Only rapid spills on land and in water were considered. Slow, hose-type leaks release small amounts of insecticide and can be controlled quickly by project personnel. As a result, the potential for exposure to such spills is much lower than that possible with larger spills. For this analysis, it is assumed that occupational exposure is the same for large or small spills since mixer/loader/drivers would be involved with the initial containment and clean-up. Furthermore, it is assumed that potential exposure to bystanders from such spills would not exceed that of workers initially involved in the cleanup (without protective equipment).

Since no data are available on occupational exposure resulting from a transportation spill, it is assumed that the total mixer/loader exposure is equivalent to 1 gallon of either the concentrated (carbaryl, trichlorfon) or diluted (acephate, diflubenzuron) insecticide in a day. Consequently, a 70 kg worker will receive a dermal exposure of 6490 mg/kg (454 g/lb x 1,000 mg/g x 1/70 kg) for each pound a.i./gallon in the mixing tank.

If the spill goes into either of the previously mentioned streams, the MC(24) would be 1,480 ppb for the 25-foot stream and 5,286 ppb for the 15-foot streams. The exposure to an individual from drinking 2 liters of this water is 0.042 and 0.151 mg/kg, respectively (1.480 or 5.286 mg/liter x 2 liter/person/day x 1 day x person/70 kg) for 1.0 lb a.i./gal mixtures. An actual spill of 1,890 gallons into a stream in eastern Oregon of carbaryl (4 lb/gal) mixed 1 to 1 with fuel oil resulted in carbaryl concentrations that ranged from 4.4 to 39.9 ppm, at the site the day of the spill (USDA Forest Service 1983). Carbaryl concentrations dropped about 5 fold within the first 24 hour period to an average residue of 8.1 ppm.

This 24 hour average is comparable to the calculated residue of 5.286 ppb which would translate to 10.5 ppm for a 2 lb/gal mixture.

The basic dose for truck spills must be adjusted for each insecticide to reflect the application rates in gypsy moth projects. The adjustment for each insecticide is as follows:

acephate - 1.5 (based upon 0.75 lb a.i./acre in 0.5 gal. mix)

carbaryl - 4.0 (Sevin 4 Oil = 4 lb a.i./gallon)

diflubenzuron - 0.06 (based upon 0.06 lb a.i./acre in 1.0 gal. mix)

trichlorfon - 1.5 (Dylox 1.5 Oil = 1.5 lb a.i.,/gallon)

A summary of the generated basic doses by accident scenario and insecticide are presented in Table 5. The calculated doses in Table 5 are for those exposure scenarios developed in the previous section, but modified for the specific insecticide application rate (a.i./acre) and mixing rate (a.i./gal).

Probability of Major Accidents Occurring

Major accidents that result in the release of insecticide can occur on gypsy moth projects. Historical records were used to develop probabilities of occurrence. Two scenarios were identified and probabilities were generated for subsequent insecticide releases (20 gallons or more) on land and in water.

Aircraft spills.--There have been no recorded incidents of aircraft spills in gypsy moth projects, so historical records for other forest insect spraying were searched. A review of Forest Service spray incident reports for spruce budworm projects in Maine (1979) and Oregon and Washington (1958-1983) indicated that 16 jettisons of formulated insecticide occurred during those years. Total acreage treated during that

time was more than 8.4 million acres. Assuming that an aircraft can treat 300 acres with each full insecticide load, it is calculated that 28,000 individual insecticide loads were required to treat 8.4 million acres (8,400,000 acres/300 acres per aircraft-load). The probability of an aircraft spill occurring can be calculated:

P_a = 16 accidents/28,000 aircraft loads
= 5.7×10^{-4} or less than 1 chance in 1000 that an aircraft spill will occur.

Where P_a = Probability of an aircraft spill per each 300 acre insecticide load.

It was assumed that 90 percent of the time application aircraft fly over land and only 10 percent of the time over small streams and ponds that a pilot cannot see from the air nor avoid. Probabilities for occurrence of aircraft insecticide spills over land and water are calculated:

$P_{al} = 0.9 \times 5.7 \times 10^{-4} = 5.1 \times 10^{-4}$ per insecticide load

$P_{aw} = 0.1 \times 5.7 \times 10^{-4} = 5.7 \times 10^{-5}$ per insecticide load

Where P_{al} and P_{aw} = Probability of an aircraft spill on land and in water, respectively, per each 300 acre insecticide load.

Truck spills.--Calculations of the probability for the occurrence of vehicular accidents in which a major spill of diluted or concentrated insecticide is released in water or on land are based upon accident rates for single unit trucks, the types commonly used in gypsy moth projects, as opposed to larger tractor-trailer or tandem trucks.

According to the Department of Transportation's Highway Statistics Division, single unit trucks, the vehicles under consideration, travelled 353,978 million miles in 1981. National Accident Sampling System statistics estimate that single unit trucks were involved in 162,000 accidents that year, or one accident for 2,185,049 miles travelled (353,978

million miles/162,000 accidents). The mean probability of a single unit truck accident can be calculated:

P_a = 1 mile/2,185,049 miles per accident = 0.000000457 or 4.6×10^{-7} per mile

Where P_a = the mean probability of a single unit truck accident per mile.

The frequency of accidents differs according to road type. The mean probability of single unit truck accidents can subsequently be adjusted to take road type into account. The following tabulation gives total miles traveled, number of accidents, and accident frequency (miles traveled per accident) for single unit truck accidents for road type based upon 1981 data. The probability of an accident occurring per mile is calculated by dividing 1 mile by the accident frequency.

	Single Unit Truck		Accident Frequency (miles	Probability of
Road Type	Total Miles (million)	Number of Accidents	traveled per accident)	Accident/Mile
Urban interstate	23,059	13,449	1,714,551	5.8×10^{-7}
Rural interstate	28,758	958	30,018,789	3.3×10^{-8}
Other urban road	146,195	92,430	1,581,683	6.3×10^{-7}

It is estimated that single unit trucks used on gypsy moth projects traveled all road types in these proportions:

Urban interstate - 30 percent.
Rural interstate - 50 percent.
Other urban roads - 20 percent.

By applying the accident probabilities for road type just generated to the proportions travelled during gypsy moth projects, an adjusted probability of occurrence for single-unit truck accidents can be calculated as follows:

$$P_a = (.30 \times 5.8 \times 10^{-7} + .50 \times 3.3 \times 10^{-8} + .20 \times 6.3 \times 10^{-7})$$
$$= 0.000000316 \text{ or } 3.2 \times 10^{-7} \text{ per mile}$$

Where P_a = Probability of an accident involving a single-unit truck occurring per mile traveled.

Not all accidents result in the release of insecticide. In estimating the potential for insecticide release, accident severity must be taken into account. As noted earlier, accident estimates provided thus far include all accidents reported to authorities regardless of severity. In adjusting for probability of insecticide release, it is assumed that only those accidents severe enough to require towing of vehicles from the scene of an accident result in the release of insecticide.

The only data base available aggregates single and tandem trucks together although size of the load is categorized. For these vehicles, 68 percent were involved in collisions with other vehicles, 21 percent with fixed objects, and 10 percent were noncollision accidents. Towing was required in 20 percent of the multi-vehicle collisions, 60 percent of the collisions with fixed objects, and 100 percent of the turnovers and ruptures.

The probability of a truck accident resulting in insecticide release can be calculated for each accident type:

$$P = P_a \times A_t \times P_t$$

Where P_a = Probability of an accident occurring per mile traveled (3.2×10^{-7})

A_t = Proportion of accidents by accident type (0.68, 0.21, and 0.10)

P_t = Proportion of accidents by accident type that require towing (0.2, 0.6, and 1.0)

For example, for accidents that involve collision with another vehicle, this computes as:

$$P = (3.2 \times 10^{-7}) \times (0.68) \times (0.2) = 4.3 \times 10^{-8}$$

The probability of insecticide release for all accident types is summarized below:

Accident Type	Probability of Release
Collision with vehicle	$p = 4.3 \times 10^{-8}$
Collision with fixed object	$p = 4.0 \times 10^{-8}$
Noncollision accident	$p = 3.2 \times 10^{-8}$
Total	$p = 1.2 \times 10^{-7}$

The probability of a truck accident releasing insecticide for all accident types is the sum of the individual probabilities or, $P = 1.2 \times 10^{-7}$ per mile traveled.

Assuming that a vehicle carrying insecticide travels 100 miles during the course of a project, the probability that a traffic accident would occur in which insecticide is spilled would be $1.2 \times 10^{-7} \times 100$ miles or 1.2×10^{-5}. It was assumed that the spill would occur on land 90 percent of the time, and in water 10 percent of the time. For this analysis, the probabilities of occurrence for a truck accident resulting in the release of insecticide are:

$P_l = 0.9 \times 1.2 \times 10^{-5} = 1.08 \times 10^{-5}$ or about 1 chance in 100,000

where P_l = probability of occurrence on land per 100 mile trip

$P_w = 0.1 \times 1.2 \times 10^{-5} = 1.2 \times 10^{-6}$ or about 1 chance in 1,000,000

where P_w = probability of occurrence in water per 100 mile trip

Summary of Probabilities for Truck and Aircraft Spills

For this analysis, the following probabilities for the occurrence of aircraft and vehicular accidents that result in the release of insecticide have been calculated.

Accident Type	Probability of Occurrence	
	On Land	In Water
Aircraft spills	5.1×10^{-4}	5.7×10^{-5}
Truck spills	1.08×10^{-5}	1.2×10^{-6}

The actual number of truck trips that are required during the course of a project depends upon the specific insecticide being used and the application rate of the insecticide. Similarly, the number of acres that a 2,000-gallon truck load of insecticide would provide coverage for is also dependent upon the particular chemical and its application rate. The following tabulation shows the number of acres that can be treated with a single 2,000-gallon truck shipment based upon the insecticide formulation and application rate.

Chemical	Application Rate	Formulation Weight Shipped	Coverage
	(lb a.i./acre)	(lbs a.i./gallon)	(acres/2,000-gallon load)
Acephate	0.75	1.5 (premixed) 1/	4,000
Trichlorfon	1.00	1.5	3,000
Carbaryl	1.00	4.0 (Sevin 4 Oil)	8,000
Diflubenzuron	0.06	0.06 (premixed) 1/	2,000

1/ Acephate and diflubenzuron are shipped as dry powders and are mixed with the desired quantity of water, usually at the aircraft loading site. For this analysis, it is assumed that these insecticides would be premixed and ready for application at the rate of 0.5 gallons/acre (acephate) and 1.0 gallon/acre (diflubenzuron).

The total number of truck trips required can be computed for any size project as can a probability of occurrence for truck spills that is specific to that project. For example, if 100,000 acres are to be treated with Sevin 4 Oil applied at the rate of 1.0 lb. a.i. per acre, the total insecticide needs could be met with 12.5 tank truck loads (100,000 acres/8,000 acres per 2,000-gallon truck load). Given the number of truck trips required, the probability of occurrence of a truck accident that involves the release of insecticide on land or in water can be calculated by multiplying the number of tank truck trips required (12.5) times the probability of occurrence on land or in water (1.08×10^{-5} or 1.2×10^{-6}, respectively). Therefore, the probability of occurrence of a truck spill occurring on land during a 100,000 acre project using carbaryl is 1.4×10^{-4} (12.5 trips x 1.08×10^{-5} per trip) or 1 truck spill for every 7,000 projects of 100,000 acres each. Table 6 presents a summary of the probabilities for accidental tank truck spills on land and in water for each insecticide based upon a 100,000-acre project.

In a manner similar to truck spills, the total number of aircraft flights required can be computed for any size project as can a probability of occurrence for aircraft-related spills that is specific to that project. The number of aircraft flights required to treat 100,000 acres can be computed by dividing the project acreage by the total acres that can be treated with each aircraft load. For example, carbaryl (Sevin 4 Oil) is applied at the rate of 0.375 gallons of mix per acre. A plane that carries 300 gallons can therefore treat 800 acres per load (300 gallons/0.375 gallons per acre). Total aircraft flights needed to treat 100,000 acres is: 100,000 acres/800 acres per load = 125 aircraft loads. The probability of an aircraft spill occurring on land or in water can be calculated by multiplying the number of aircraft loads (125) times the probability of occurrence on land or in water (5.1×10^{-4} or 5.7×10^{-5}, respectively). Therefore, the probability of an aircraft spill occurring on land during a 100,000-acre project using carbaryl is 6.3×10^{-2} (125 aircraft loads x 5.1×10^{-4} per load) or 1 aircraft spill for every 16 projects of 100,000 acres each. Probabilities for aircraft spills on land and in water for each insecticide, based upon a 100,000-acre project, are summarized in Table 6.

Frequency of Exposure During a Lifetime

In order to calculate the probability of long-term health effects, it is necessary to estimate the frequency, or the number of times, that an individual could be exposed to insecticides used in suppression or eradication projects over a lifetime. This requires estimates of: 1) an average life span; 2) the number of times that a suppression or eradication project could take place on the same specific acres in a lifetime; and 3) the number of applications of insecticides and hence the number of possible exposures to an individual over a lifetime. An exposure event in a lifetime includes: 1) exposure from the direct application, 2) secondary exposure or contact with spray residues on grass, foliage, cars, etc., and 3) eating or drinking contaminated foods.

The average life span of an individual is assumed to be 70 years. Estimates of the number of suppression or eradication projects that could occur over the 70-year lifetime of an individual require several assumptions that recognize the difference in objectives as well as geography between the two types of projects.

Gypsy moth eradication projects are conducted in areas of the country where infestations have been established by artificial means (i.e., household moves, campers, etc.) as opposed to natural spread. For the purpose of this analysis, it is assumed that only eradication projects will be conducted in these areas over a 70-year period. It is also possible that future applications during the 70-year period may involve implementation of other techniques besides or in place of chemical insecticides. Therefore, frequencies were calculated for the chemical insecticides as a group; the worst case assumption being that during the 70-year period an individual(s) will be exposed to only chemical insecticides in eradication projects. Up to 3 applications of chemical insecticides may be applied over a 6-week period in order to achieve eradication goals. It is probable that the insect could be artificially re-introduced in the same acreage one more time in a 70-year period. Therefore, the frequency of exposure to an individual living on that acreage over a 70-year lifetime is estimated to be:

3 exposures/project x 2 projects/lifetime = 6 exposures/lifetime

Suppression projects are usually conducted only in areas of the country where gypsy moth is firmly established, spreading naturally and exhibiting normal cyclic outbreaks. For this analysis, the basic assumptions were similar to those described above. Only suppression activities will be conducted in the area over a 70-year period, and generation of the exposure frequencies will be based upon use of chemical insecticides. When these insecticides are used in suppression projects, there is one application per project. For this analysis, it is estimated that on the average, over a 70-year lifetime, a suppression project could realistically be conducted in the same area every 7 years or 10 times per lifetime. The frequency of exposure to an individual from chemical insecticides over a 70-year lifetime is estimated to be:

1 exposure/project x 10 projects/lifetime = 10 exposures/lifetime

Population at Risk

Public at Large

Two separate populations can be exposed to the chemical insecticides, the general public and workers. The general public is considered to live in the same area for a 70-year lifetime (or if moving to another area, receive a similar worst case exposure over a lifetime), and is exposed to the maximum calculated dose. The hypothetical maximum exposed individual weighs 70 kg and ingests 2 liters of water per day and 0.5 kg/day of each item of food. This overestimates the average daily consumption of food.

For urban/suburban areas, a density of four houses per acre with four members of a family per household results in 16 people/acre. This may be compared with an average density for metropolitan area from 1980 census data:

Total metropolitan population	= 167 million people
Total metropolitan areas	= 47.4 million acres
Average metropolitan population density	= 3.52 people/acre

Total rural population	= 59.5 million people
Total rural areas	= 2.21 billion acres
Average rural population density	= 0.03 people/acre

For the purpose of this analysis, we have arbitrarily increased the value of the rural population density from 0.03 to 0.2 people/acre. The census figures take into consideration vast areas of sparsely populated agricultural and prairie grass lands in the central portion of the country. Rural areas, for the purposes of this analysis, are more typically like those found in the eastern United States, the Lake States, and along the western coast. Postulating an average farm size of 200 acres with a family of 4, the value becomes 0.2 people/acre.

For gypsy moth eradication projects, the approximate historic breakdown of target areas, based upon location of treatments from 1967-1983 is:

Area	Percent of Program	Population Density per Acre
Urban/suburban	59	16
Rural	41	0.2

A composite population density is: (0.59 x 16) + (0.41 x 0.2) = 9.5 people/acre.

For gypsy moth suppression projects, the general historic breakdown of target areas is:

Area	Percent of Program	Population Density per Acre
Urban/suburban	85	16
Rural	15	0.2

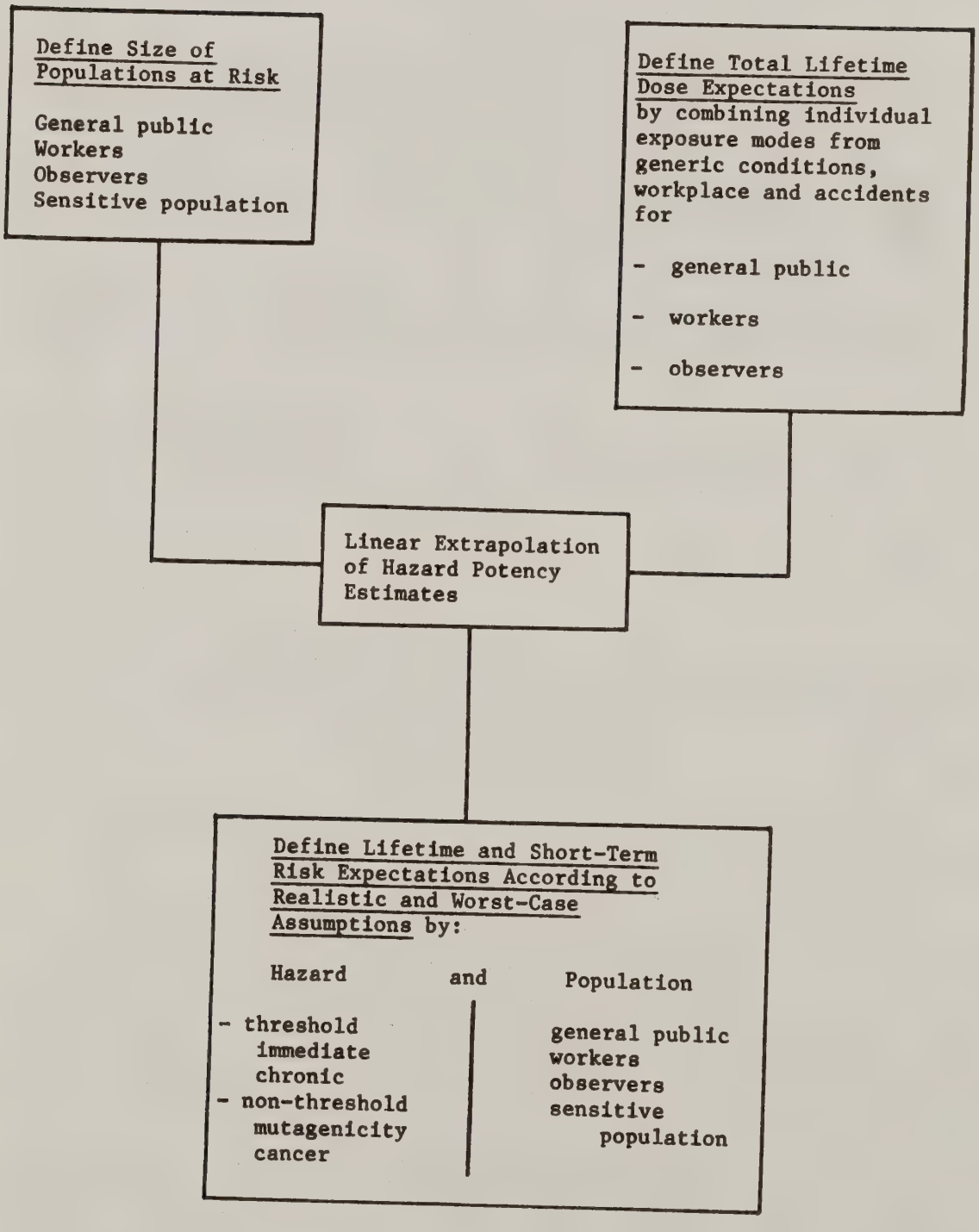
A composite population density for gypsy moth suppression projects is:

(0.85 x 16) + (0.15 x 0.2) = 13.6 people/acre

For this analysis, a conservative estimate of 14 people/acre will be used for both eradication and suppression projects.

As indicated by the exposure data generated earlier in this analysis, the dose potential is greatest for the occupationally exposed group. One can assume that those residents who remain indoors during spraying do not receive any direct doses, but may pick up some contamination afterwards when walking outside. If one assumed that half the population remains indoors (most spraying occurs in early morning, although in some cases, spraying conditions could last through early afternoon), then only one-half of the population would receive a full, direct maximum dose. All of those outdoors in the spray area and observing the treatment, receive the maximum dose and are therefore considered as maximum exposed individuals. Those downwind receive proportionally less as described earlier.

EVALUATION OF RISK



EVALUATION OF RISK

A summary of established exposure thresholds, ADI, and carcinogenic potency slopes used in the analysis is presented in Table 7. The NOELs selected represent the lowest and next lowest values identified in Tables 1 through 4. The ADI's are empirically derived values that combine maximum no-observed adverse-effect levels with uncertainty (safety) factors. The uncertainty factors used to calculate the ADIs represent the level of confidence in the animal and human toxicity data (NAS-NRC 1983). Therefore the ADI, being an internationally recognized concept for evaluating human exposure to pesticides, is an acceptable standard for evaluating overall human health risks because it is based on the evaluation and significance of the total data base.

A brief discussion of how the ADIs were derived for acephate, carbaryl, diflubenzuron, and trichlorfon provides some insight into the types of safety factors used and which are internationally accepted. The ADI for acephate (0.025 mg/kg/day) was established by EPA and is based on the 28 month rat feeding/oncogenic study with a NOEL of 0.25 mg/kg/day and a safety factor of 10 (USEPA 1982a). The ADI for carbaryl (0.1 mg/kg/day) was established by EPA and is based upon a 2 year rat feeding study with a NOEL of 10 mg/kg/day and a safety factor of 100 for extrapolation from animal to man (Dr. Jeffrey Charles, Union Carbide Corporation, personal communication). The ADI for diflubenzuron (0.011 mg/kg/day) was established by EPA and is based upon a NOEL of 1.1 mg/kg/day which was determined by regression analysis of the 13 and 80 week mouse feeding studies (see EPA comment letter #29, Appendix G). The ADI thus reflects a safety factor of 100. The ADI for diflubenzuron is provisional pending review of a carcinogenicity study (the review was recently completed). A provisional ADI is generally derived using a higher safety factor to account for needed data. The ADI for trichlorfon (0.01 mg/kg/day) was established by WHO (Mobay Undated). The NOEL and safety factor used to derive this ADI are not identified; however, the lowest NOEL listed in

Table 4 (1.25 mg/kg/day) suggests a safety factor of 100. EPA currently rates the study supporting this NOEL as supplemental, meaning that the information is useful but the study no longer meets current protocols. Since this was the lowest NOEL value found for trichlorfon, it was included to ensure that risks would not be underestimated.

ADI and the lowest NOEL identified for each insecticide are compared to the realistic and worst case doses in order to evaluate the risks for nongenetic responses such as teratogenicity (birth defects), fetotoxicity, or systemic toxicity (such as liver dysfunction, reduced fetus and organ weight, food consumption, or blood chemistry). This provides a straight forward method for evaluating the overall health risks. For example, for any dose that is equal to or below the ADI, it can be said that the dose is within an acceptable margin of safety (Doull et al. 1980). Where realistic and worst case doses exceed the ADI a comparison with the NOEL is made. Except for carbaryl, where the lowest NOEL is for teratogenicity in dogs, the lowest NOELs are for either cholinesterase depression or systemic effects. These are characteristic low dose responses for the organophosphate insecticides, acephate and trichlorfon. The teratogenicity NOELs for acephate, trichlorfon, and diflubenzuron are at least an order of magnitude higher than the lowest NOEL used to compare to the realistic and worst case doses.

General public exposure scenarios include exposures that result from the direct application of the insecticide, and a combination of direct application and secondary exposures that result from residues that might be in water or food (dietary). Tables 8 through 11 compare various degrees of exposure for both the general public and those persons involved with the application of the insecticide. These are then separated into groups of individuals who could receive either a maximum realistic dose or those who could receive a worst case dose through abnormal variations in insecticide application.

The calculated realistic and worst case doses are compared to the lowest NOEL or ADI by calculating the ratio of NOEL/dose and ADI/dose. This ratio is sometimes referred to as the margin-of-safety (MOS) (Witt 1984). If the dose is greater than the NOEL or ADI, the comparison is made by determining the inverse ratio (i.e., dose/NOEL). In Tables 8 through 15, these calculated ratios are expressed as positive or negative numbers. A positive number indicates the number of times those values are below the established ADI, NOEL, or LD_{50}. A negative number indicates the number of times these values are above the ADI, NOEL, or LD_{50}. For example, when the realistic dose for an individual directly exposed to acephate (0.0017 mg/kg/day) is compared to the ADI and the lowest NOEL (Table 7), the ratios are approximately 15 and 147. Therefore, the dose received is 15 times below the ADI and 147 times below the NOEL.

The accident exposure scenarios in Tables 12 through 15, evaluate the effects of a one time exposure of relatively short duration to higher dose levels than would normally occur during insecticide application. The realistic and worst case doses for the accident scenarios are calculated in the same manner as the realistic and worst case doses for the general population and for occupational scenarios. These estimated doses are compared to the acute dermal LD_{50} (for exposure via direct contact), the ADI, and the lowest NOEL (for exposure via consumption of contaminated water) by calculating the ratio of the LD_{50}/dose, NOEL/dose or ADI/dose. If the dose is greater than the LD_{50}, NOEL or ADI, the comparison is made by determining the inverse ratio as previously described. It is important to note that the dermal LD_{50} values listed in Table 7 actually represent the highest doses tested without causing fatalities from dermal exposure.

For genetic responses, the cancer potency along with the expected average lifetime dose over a 70-year period was used to evaluate the risk of cancer incidences for N-nitrosocarbaryl and trichlorfon. The qualitative risk of heritable mutations is based on the overall evidence of whether or not the insecticides are mutagenic in humans.

General Public

Acephate.--The expected doses and comparison to established thresholds for acephate are presented in Table 8. The doses identified for the general public and occupationally exposed individuals are all below the lowest NOEL established for acephate by factors ranging from about 2 to 758. The worst case doses for those individuals in the general public which combine exposure to the insecticide and consumption of food containing acephate residues are higher than the established ADI. The remaining doses calculated for the general public are equivalent to or below the ADI. The mixer/loader occupational group could receive the highest dose, estimated at 6 times above the ADI, but still 2 times below the lowest established NOEL. This dose is 67 times less than the highest dose tested (without effect), in teratogenicity studies. The realistic and worst case doses in water that result from insecticide spills (aircraft and truck accidents) range from 8 times below, to 2 times above the lowest NOEL (cholinesterase inhibition). The realistic dose resulting from aircraft spills in water is approximately equal to the ADI, while the worst case dose is slightly above the ADI. Truck spills in water exceed the ADI by 10 to 18 times.

Dermal exposure levels from accidental spills are presented in Table 12. Dermal exposures received from aircraft spills result in doses that range from more than 47 to 177 times below the highest exposure levels tested (with no effect) for the LD_{50}. The number of people that could be dermally exposed to an aircraft spill is estimated to be 35. The probability of an aircraft accident resulting in an insecticide spill on land is calculated to be 5.1×10^{-4} or 1 aircraft spill for every 1,960 aircraft flights. If an aircraft spill occurs, the probability that it contains a worst case dose is 1.8×10^{-3} or, for every 556 spills, 1 spill would contain a worst case dose. In other words, the probability of an aircraft accident occurring in association with a worst case dose is 0.9×10^{-6} or about 1 chance in a million.

Dermal exposures received from a tank truck accident are up to twice as high as the highest exposure level tested (with no effect) for the dermal LD_{50}. The probability of a truck accident occurring and the insecticide being released on land is calculated to be 1.08×10^{-5} or 1 insecticide spill for every 93,000 vehicle trips. The number of people that could be exposed to a tank truck spill is variable and is not calculated; however, the health risk associated with exposure to such a spill is discussed later in this analysis. The probability of a truck spill occurring in association with a worst case dose is 1.9×10^{-8} or about 1 chance in 50 million.

Carbaryl.--The expected doses and comparisons to established thresholds are presented in Table 9. The doses identified for the general public and occupationally exposed individuals are all below the lowest NOEL established for carbaryl by factors ranging from 16 to 7,102. All doses are below the ADI except for the worst case doses for the mixer/loader group and the worst case doses for the general public which include a dietary component. In these cases the doses range up to 2 times more than the ADI. The worst case dose received by the highest exposed group (mixer/loaders) is 16 times lower than the lowest teratogenic NOEL for carbaryl.

The realistic and worst cases doses in water that result from insecticide spills (aircraft and vehicle accidents) range from 5 to 41 times below the lowest NOEL (Table 13). Only doses associated with truck spills in water are above the ADI. The probability of a truck spill contaminating water is estimated to be 1.2×10^{-6} or 1 spill for every 833,333 vehicle trips.

All dermal exposures received from aircraft related spills range from 20 to 77 times below the highest exposure level tested (with no effect) for the dermal LD_{50} (Table 13). The population exposed to an aircraft spill is estimated to be 35 people. The probability of an aircraft related insecticide spill occurring is calculated to be 5.1×10^{-4} or 1 aircraft-related spill for every 1,960 aircraft flights. If an aircraft spill occurs, the probability that it contains a worst case dose is 1.8×10^{-3} or, for every 556 spills, 1 spill would contain a worst case dose.

The probability of an aircraft spill occurring in association with a worst case dose is the product of these probabilities (0.9×10^{-6}) or about 1 chance in a million. All of the dermal exposures received from contact with concentrated carbaryl spills (truck spills) are calculated to be 5 times above the highest dose tested without reaching a dermal LD_{50}. The probability of a truck accident occurring and insecticide being released is calculated to be 1.08×10^{-5} or 1 insecticide spill for every 93,000 vehicle trips. The number of individuals that could be exposed to a tank truck spill is variable and is not calculated; however, the health risk associated with exposure to such a spill is discussed.

Diflubenzuron.--The expected doses and comparisons to established thresholds for diflubenzuron are presented in Table 10. The realistic and worst case doses for the general public and occupationally exposured individuals range from 92 to more than 42,000 times below the lowest NOEL which is for MHb (methemoglobin) and SHb (sulfhemoglobin). These doses are equal to or more than 400 times below the ADI. The worst case dose received by the group with the highest exposure (mixer/loader) is more than 300,000 times below the lowest teratogenic NOEL for diflubenzuron listed in Table 7.

Dermal exposures resulting from all accident types (Table 14) range from 3 to more than 870 times less than the highest exposure tested (with no effect) for the dermal LD_{50}. Doses received from consumption of water into which diflubenzuron has spilled are 61 to 733 times below the lowest NOEL listed in Table 7. The probabilities of aircraft and vehicular spills (for realistic and worst case doses) occurring, and the estimated number of people exposed to these accidents are the same as those described previously for acephate and carbaryl.

Trichlorfon.--The expected doses and comparisons to established thresholds for trichlorfon are presented in Table 11. The realistic and worst case doses for the general public and occupationally exposured individuals range from 5 to 2,273 times below the lowest NOEL. The same doses range from 23 times below the ADI to 20 times above the ADI. All but

one of the doses above the ADI occur for worst case exposures to occupationally exposed individuals and general public scenarios that include a dietary component. That exception above the ADI occurs for realistic exposures in the mixer/loader group. The worst case dose received by the group with the highest exposure (mixer/loaders) is 40 times below the lowest teratogenic NOEL listed for trichlorfon in Table 7.

The doses received from consumption of water containing trichlorfon residues (from accidental spills) range from 4 to 30 times below the lowest NOEL (Table 15). All doses are above the ADI. Dermal exposure levels from accidental spills are also presented in Table 15. Dermal exposures from aircraft spills are more than 10 to 36 times below the highest exposure level tested (with no effect) for the dermal LD_{50}. Dermal exposures from insecticide spills (truck accidents) could exceed by 5 times the exposure level tested (with no effect) for the dermal LD_{50}. The probabilities of aircraft and vehicular spills (for realistic and worst case doses) occurring, and the estimated number of people exposed to these accidents are the same as those described previously for acephate and carbaryl.

Nonthreshold Responses

Assessment of Cancer Risk

The expected probability of cancer caused by exposure to carbaryl or trichlorfon (realistic or worst case dose) used in gypsy moth eradication or suppression projects is determined using the following equation:

$$R = \beta_h d$$

Where β_h is the cancer potency for humans in $(mg/kg/day)^{-1}$ and d is the lifetime average dose, (mg/kg/day) (Crouch and Wilson 1979 and Crouch et al. 1983).

Since exposure to carbaryl or trichlorfon resulting from their use in gypsy moth eradication or suppression projects does not occur daily over a 70-year lifetime, lifetime average dose must be calculated based on the number of projects, number of days exposure during each project, and amount of exposure during each of those days. Exposure and the resulting dose to the general public varies depending on whether individuals are directly exposed during application, stay inside and receive only indirect exposure, or eat contaminated meats or vegetables. The persistence of the insecticide is also a factor when considering indirect exposure from eating contaminated produce or from rubbing surfaces that have spray residues on them. Average lifetime dose (d) is calculated as follows:

$$d = (d_i + d_1 + d_2 + \ldots d_f) \times E/25{,}550$$

Where d_i = dose received the day of spray
d_1 = indirect dose 1 day after spray
d_2, d_3 ... d_f = indirect dose levels 2, 3 or f days (insecticide dissipates) after spray
E = number of applications per lifetime (either 6 for eradication or 10 for suppression projects)
25,500 = number of days in a 70-year lifetime

N-nitrosocarbaryl.--For carbaryl, the only dose that needs to be considered is the oral or dietary dose because the concern is with the possible reaction of nitrite ions and carbaryl in the stomach. This reaction will not take place under the conditions found on the skin. The N-nitrosocarbaryl dose can be calculated from carbaryl doses as follows:

N-nitrosocarbaryl dose = carbaryl dose x (230/201) x 0.002

Where:

(230/201) = molecular weight of N-nitrosocarbaryl divided by the molecular weight of carbaryl, which assumes that all of the carbaryl would be converted to nitrosocarbaryl.

0.002 = the *in vivo* yield compared to theoretical yield (Rickard and Dorough 1979).

For example, a realistic oral (dietary) dose of carbaryl of 0.0094 mg/kg/day (Table 9) represents a hypothetical N-nitrosocarbaryl dose of 0.000022 mg/kg/day (0.0094 mg/kg/day x 230/201 x .002) which represents the d_i or the initial dose.

Dose levels for subsequent days (d_1 through d_f) are lower because of the degradation of the carbaryl residue in meat, water, or vegetables. As determined earlier, meat residues decline to zero in 7 days, vegetables in 14 days, and water in 4 days. Degradation was assumed to be linear over the longest time period and the degradation period was broken into 3 equal parts with average doses of 3/4, 1/2, and 1/4 the initial dose level. Therefore, the total dietary dose of carbaryl will degrade over 14 days, with days 1 through 4 having an average dose of 0.007 mg/kg/day (0.0094 mg/kg/day x 0.75), days 5 to 9 having an average dose of 0.0047 mg/kg/day (0.0094 mg/kg/day x 0.5), and days 9 to 14 having an average dose of 0.0024 mg/kg/day (0.0094 mg/kg/day x 0.25). These carbaryl doses translate to N-nitrosocarbaryl dose of 0.000013 mg/kg/day, 0.0000087 mg/kg/day, and 0.0000044 mg/kg/day. The realistic lifetime dose of N-nitrosocarbaryl from eradication projects is then:

$$d = (0.000022 \text{ mg/kg/day} + 0.00006 \text{ mg/kg/day} \times 4 \text{ days} + 0.000011 \text{ mg/kg/day} \times 4 \text{ days} + 0.0000054 \text{ mg/kg/day} \times 4 \text{ days}) \times 6 \text{ projects/lifetime}/25{,}550 \text{ days/lifetime}$$

$$d = 3.56 \times 10^{-8} \text{ mg/kg/day}$$

The worst case lifetime dietary dose is calculated in a similar manner. The carbaryl dose at the day of spraying, d_i, is 0.116 mg/kg/day (Table 9). Doses for the remaining 13 days are 0.087 mg/kg/day (0.116 mg/kg/day x 0.75) for days 1 to 5, 0.058 mg/kg/day (0.116 mg/kg/day x 0.5) for days 5 to 9, and 0.029 mg/kg/day (0.116 mg/kg/day x 0.25) for days 9 to 14. N-nitrosocarbaryl doses are 0.00027, 0.00020, 0.00014, and 0.000067 mg/kg/day, respectively. Worst case lifetime dose of N-nitrosocarbaryl from eradication projects is calculated as follows:

$$d = (0.00027 \text{ mg/kg/day} + 0.00020 \text{ mg/kg/day} \times 4 \text{ days} + 0.00014 \text{ mg/kg/day} \times 4 \text{ days} + 0.000067 \text{ mg/kg/day} \times 4 \text{ days}) \times 6 \text{ projects/lifetime} / 25{,}550 \text{ days/lifetime}$$

$$d = 4.45 \times 10^{-7} \text{ mg/kg/day}$$

Average lifetime doses for suppression projects are calculated by multiplying the eradication doses times 1.67 (the ratio of 10 projects/6 projects), or 5.94×10^{-8} and 7.43×10^{-7} mg/kg/day for realistic and worst case, respectively.

The cancer risk to an individual exposed to realistic or worst case dietary doses of carbaryl for eradication is then calculated as follows:

$$R \text{ (risk)} = \beta_1 d = 0.057 \text{ (mg/kg/day)}^{-1} \times d$$

For a realistic dose:

$$R = 0.057 \text{ (mg/kg/day)}^{-1} \times 3.56 \times 10^{-8} \text{ mg/kg/day}$$
$$= 2.03 \times 10^{-9} \text{ or a risk of about two in a billion.}$$

For worst case:

$$R = 0.057 \text{ (mg/kg/day)}^{-1} \times 4.45 \times 10^{-7} \text{ mg/kg/day}$$
$$= 2.54 \times 10^{-8} \text{ or a risk of about two in 100 million.}$$

Cancer risks to an individual exposed to realistic or worst case dietary doses of carbaryl resulting from suppression projects are 3.38×10^{-9} and 4.23×10^{-8}, respectively.

Trichlorfon.--The cancer risk from exposure to trichlorfon was calculated only for the highest two exposure groups: (1) residents who receive direct exposure to insecticide during treatment and who eat contaminated food and drink contaminated water, and (2) workers (observers) who are outside and receive a direct application and who then eat

contaminated food and water. Groups with lower exposures will have cancer risks that are lower than these two highest exposed groups. Average lifetime dose (d) is based on two events: initial dose resulting from the application, and secondary exposures to residues that degrade over time. The initial doses (in mg/kg/day) for d_1, (Table 11) are: 0.012 for realistic direct and dietary; 0.126 for worst case direct and dietary; 0.012 for realistic observer and dietary; and 0.174 for worst case observer and dietary.

Initial doses resulting from exposure to dislodgable residues are equal to the indirect dose. These are 0.0004 mg/kg/day for realistic and 0.0009 mg/kg/day for worst case doses (Table 11). Total initial secondary dose used in the calculation is the sum of dietary and indirect doses. These dose values are 0.00984 mg/kg/day for realistic doses and 0.1178 mg/kg/day for worst case doses. For realistic doses, these residues were assumed to degrade at the same rate as those found on vegetables, or decrease to zero over a 2-week time period. Therefore, daily doses from diet and dislodgable residues can be calculated using the same linear assumptions for degradation as was used for carbaryl. Realistic doses for residues that are dislodgable after application are 0.0074 mg/kg/day (0.00984 mg/kg/day x 0.75) for days 1 to 5, 0.0049 mg/kg/day (0.00984 mg/kg/day x 0.5) for days 5 to 9, and 0.0025 mg/kg/day (0.00984 mg/kg/day x 0.25) for days 9 to 14.

For the worst case, dislodgable residues are degraded over a 60-day time period (FEIS, p. 65). Degradation over the first 14 days follows the same linear assumption for degradation used for the realistic dose. These doses are 0.088 mg/kg/day for days 1 to 5, 0.059 mg/kg/day for days 5 to 9, and 0.029 mg/kg/day for days 9 to 14. The day 15 to day 60 time period is similarly broken into 3 equal parts, but the initial dose was 0.00045 mg/kg/day (25 percent of the initial dislodgable level of 0.0018 mg/kg/day). Therefore, worst case dose levels for days 15 to 30 are 0.00034 mg/kg/day (0.00045 mg/kg/day x 0.75), 0.00022 mg/kg/day (0.00045 mg/kg/day x 0.5) for days 30 to 45, and 0.00011 mg/kg/day (0.00045 mg/kg/day x 0.25) for days 45 to 60. Average lifetime dose (d) for eradication projects are calculated as follows:

d (realistic direct plus dietary) = (0.012 mg/kg/day + 0.0074 mg/kg/day x 4 days + 0.0049 mg/kg/day) x 4 days + 0.0025 mg/kg/day x 4) x 6 projects/lifetime/25,550 days/lifetime

= 0.0000167 mg/kg/day or

= 1.67×10^{-5} mg/kg/day

d (worst case direct plus dietary) = (0.126 mg/kg/day + 0.088 mg/kg/day x 4 days + 0.059 mg/kg/day x 4 days + 0.029 mg/kg/day x 4 days + 0.00034 mg/kg/day x 15 days + 0.00022 mg/kg/day x 15 days + 0.00011 mg/kg/day x 15 days) x 6 projects/lifetime/25,550 days/lifetime

= 0.000197 mg/kg/day or

= 1.97×10^{-5} mg/kg/day

d (realistic observer plus dietary) = 1.67×10^{-5} mg/kg/day

d (worst case observer plus dietary) = (0.174 mg/kg/day + 0.088 mg/kg/day x 4 days + 0.059 mg/kg/day x 4 days + 0.029 mg/kg/day x 4 days + 0.00034 mg/kg/day x 15 days + 0.00022 mg/kg/day x 15 days + 0.00011 mg/kg/day x 15 days) x 6 projects/lifetime/25,550 days/lifetime

= 0.000209 mg/kg/day or

= 2.09×10^{-4} mg/kg/day

As before, average lifetime dose resulting from suppression projects can be calculated by multiplying the doses for eradication by 1.67. Cancer risk to an individual is calculated as follows:

$$R = \beta(\text{humans}) \times d = 0.0047\ (\text{mg/kg/day})^{-1} \times d$$

$$= 0.0047\ (\text{mg/kg/day})^{-1} \times 1.67 \times 10^{-5}\ \text{mg/kg/day (realistic dose for direct plus dietary)}$$

$$= 7.85 \times 10^{-8}$$ Risks for other trichlorfon exposures are:

Exposure	Eradication (6 applications)		Suppression (10 applications)	
	Lifetime Dose (mg/kg/day)	Lifetime Cancer Risk	Lifetime Dose (mg/kg/day)	Lifetime Cancer Risk
Realistic direct + dietary	1.67×10^{-5}	7.85×10^{-8}	2.79×10^{-5}	1.31×10^{-7}
Worst case direct + dietary	1.97×10^{-4}	9.26×10^{-7}	3.29×10^{-4}	1.55×10^{-6}
Realistic observer + dietary	1.67×10^{-5}	7.85×10^{-8}	2.79×10^{-5}	1.31×10^{-7}
Worst case observer + dietary	2.09×10^{-4}	9.82×10^{-7}	3.49×10^{-4}	1.64×10^{-6}

Acephate.--As was done with trichlorfon, the cancer risk from exposure to acephate was calculated only for the highest two exposure groups: direct plus dietary and observer plus dietary. The initial doses (in mg/kg/day) for d_i (Table 8) are: 0.025 for realistic direct and dietary, 0.11 for worst case direct and dietary, 0.026 for realistic observer and dietary, and 0.147 for worst case observer and dietary. The total initial secondary exposures used in this calculation (indirect and dietary, Table 8) are 0.024 mg/kg for realistic and 0.108 mg/kg/day for worst case. Based on the data for broccoli and lettuce (Chevron Chemical Co. 1973, also page F-43), dislodgable residues and residues in or on food were assumed to degrade to zero over a 20 day time period. Degradation was assumed to be linear. Therefore, realistic doses for residues that are dislodgable or consumed after application are 0.018 mg/kg/day for days 1 to 8, 0.012 mg/kg/day for days 8 to 14, and 0.006 mg/kg/day for days 14 to 20. Worst case residues were degraded over the same 20 day time period and were 0.081, 0.054, and 0.027 mg/kg/day respectively.

The average lifetime dose for eradication projects are calculated as follows:

d (realistic direct plus dietary) = (0.025 mg/kg/day + 0.018 mg/kg/day x 6 days + 0.012 mg/kg/day x 6 days + 0.006 mg/kg/day x 7 days) x 6 projects/lifetime/25,550 days/lifetime

= 0.000058 mg/kg/day

The increased lifetime cancer risk to an individual receiving this type of exposure is calculated as follows:

$$R = \beta \text{ (humans)} \times d$$
$$= 0.025 \text{ (mg/kg/day)}^{-1} \times 5.8 \times 10^{-5} \text{ mg/kg/day}$$
$$= 1.4 \times 10^{-6}$$

Lifetime doses and cancer risks for the other acephate exposures are:

Exposure	Eradication (6 applications)		Suppression (10 applications)	
	Lifetime dose (mg/kg/day)	Lifetime cancer risk	Lifetime dose (mg/kg/day)	Lifetime cancer risk
Realistic direct + dietary	5.8×10^{-5}	1.4×10^{-6}	9.69×10^{-5}	2.4×10^{-6}
Worst case direct + dietary	1.84×10^{-4}	4.6×10^{-6}	3.08×10^{-4}	7.7×10^{-6}
Realistic observer + dietary	5.8×10^{-5}	1.4×10^{-6}	9.69×10^{-5}	2.4×10^{-6}
Worst case observer	2.69×10^{-4}	6.7×10^{-6}	4.49×10^{-4}	1.1×10^{-5}

In order to calculate the cancer risk to an entire population, the weighted risk must be calculated, taking into account the probability of either worst case or realistic exposures. The weighted cancer risk is calculated as follows:

0.0018 x (worst case risk estimate) + 0.9982 x (realistic risk estimate)

where 0.0018 is the probability of worst case dose being applied, and 0.9982 is the probability of realistic dose being applied (see page F-51). Weighted lifetime risks are presented in Table 16.

To estimate the number of possible incidences of cancer per acre over a lifetime series of applications, the cancer risk is multiplied by the population at risk (14 individuals/acre based on assumptions stated on p. F-65). This translates to the lifetime incidences of cancer per acre for the lifetime number of applications:

Insecticide/ Exposure Scenario	Incidences of Cancer/acre/lifetime	
	Suppression (for 10 applications)	Eradication (for 6 applications)
Carbaryl		
Dietary	4.84×10^{-8}	2.90×10^{-8}
Trichlorfon		
Observer and Dietary	1.88×10^{-6}	1.12×10^{-6}
Direct and Dietary	1.88×10^{-6}	1.12×10^{-6}
Acephate		
Observer and Dietary	3.2×10^{-5}	1.97×10^{-5}
Direct and Dietary	3.2×10^{-5}	1.97×10^{-5}

In a site-specific environmental assessment, total incidences of cancer in the population can be calculated for a single application by dividing incidences of cancer/acre/lifetime by number of applications (6 or 10) and multiplying by the total number of acres proposed for treatment. For example, for suppression projects, incidents of cancer are calculated as follows (example for carbaryl: 4.84×10^{-8}/10 applications x no. of acres treated):

Carbaryl

Dietary	No. of acres x 4.84×10^{-9}

Trichlorfon

Observer and dietary	No. of acres x 1.88×10^{-7}
Direct and dietary	No. of acres x 1.88×10^{-7}

Acephate

Observer and dietary	No. of acres x 3.2×10^{-6}
Direct and dietary	No. of acres x 3.2×10^{-6}

In other words, there would be less than one incidence of cancer if carbaryl were sprayed on 100 million acres, or if trichlorfon were sprayed on a million acres, or if acephate were sprayed on 500,000 acres.

Diflubenzuron.--Because the cancer potency of diflubenzuron could be considered to be zero (based on the mouse study described on p. F-21), specific lifetime dose values were not calculated. However, because of the uncertainty about the carcinogenic potential of 4-chloroaniline, a metabolite of diflubenzuron, some cancer risk estimates used to be discussed based on the worst case assumption that 4-chloroaniline was a carcinogen (a kind of "what if" discussion). Since NCI found no conclusive evidence of carcinogenicity of 4-chloroaniline, its cancer potency would certainly be less than that calculated for acephate (0.025 kg • day/mg).

Since diflubenzuron is considered a non-carcinogen, the supposed risk would come from eating meats containing 4-chloroaniline. A review of the environmental fate data discussed in the Diflubenzuron Decision Document (USEPA 1979) shows that fish represent a worst case for meats because about 60 percent of the metabolic residues in fish meat was found to be 4-choroaniline. A rapid depletion of the residues was reported in the

Decision document but no data were given. Since diflubenzuron doses (Table 11) are about 10 to 20 times lower than those for the other 3 insecticides, and eating meats only accounts for about 8 percent of the total exposure, potential lifetime exposures to 4-chloroaniline resulting for the use of diflubenzuron would be about 1000 times lower than those for trichlorfon even if the 4-chloroaniline persisted in the meat for 60 days. Such estimated doses and the estimated cancer potency results in worst case lifetime cancer estimates in the range of $1x10^{-8}$ to $1x10^{-9}$ for individuals who may eat meat that contains 4-chloroaniline resulting from the use of diflubenzuron in gypsy moth projects.

Accidents.--Based on the cancer risk model used in this analysis, cancer could occur from a single high dose because the model is based on the theory that exposure to a single molecule could cause an incidence of cancer. As was seen in the exposure section, high doses to acephate, carbaryl, or trichlorfon would occur if an individual were exposed to an accidental spill (Tables 12, 13 and 15). Since exposures resulting from an accident are primarily dermal, N-nitrosocarbaryl cannot be formed; therefore, no risk of cancer was determined for dermal exposures. However, a cancer risk was calculated for consumption of water containing residues of carbaryl. To evaluate the risk of cancer from exposure to acephate, carbaryl, or trichlorfon resulting from an accident, the single high dermal exposure, or water consumption values, need to be expressed in terms of average lifetime dose. For example, the realistic exposure of 58 mg/kg/day (Table 15) for an individual dermally exposed to trichlorfon from an aircraft spill is converted to dose by applying the 10 percent dermal absorption factor (5.8 mg/kg/day = 58 mg/kg/day x 0.1). The average lifetime dose of 0.00023 mg/kg/day (5.8 mg/kg/day x 1 day/25,500 days) is calculated by dividing by the number of days in a 70 year lifetime. The cancer risk to an individual exposed to such a spill is:

$$R = \beta_h d = 0.0047 \text{ (mg/kg/day)}^{-1} \times 0.00023 \text{ mg/kg/day} = 1.08 \times 10^{-6}$$

For drinking water exposure equals dose, Therefore, lifetime average doses are calculated by dividing the exposure value by days in a lifetime.

It needs to be pointed out that averaging a single large dose over the lifetime of an individual introduces uncertainty into the cancer risk calculation. The cancer potency term (β) is determined from studies where animals have been given chronic doses over a time period that approaches the natural life span of the animal. In the case of a large single exposure, as would result from an accident, the dose occurs only once in the lifetime of the individual. This high dose could overwhelm the body's mechanisms to detoxify the chemical or overwhelm the DNA repair mechanisms. Thus, cancer risk would be increased. Conversely, risk could be reduced because the chemical would only be in the body a short period of time (1 day in a lifetime) compared to the dosing period which was used to calculate the cancer potency (β).

The cancer risks associated with the accident scenarios for trichlorfon and carbaryl are:

Scenario	Realistic	Worst Case
Trichlorfon		
Aircraft Spill:		
Dermal (partial)	1.08×10^{-6}	1.95×10^{-6}
Dermal (full)	2.23×10^{-6}	4.05×10^{-6}
Water (drinking)	6.08×10^{-9}	1.11×10^{-8}
Truck Spill:		
Dermal	1.87×10^{-4}	1.87×10^{-4}
Water (drinking)	4.18×10^{-8}	4.18×10^{-8}

Carbaryl

Aircraft Spill:

Water (drinking)	3.93×10^{-10}	7.15×10^{-10}

Truck Spill

Water (drinking)	3.08×10^{-9}	3.08×10^{-9}

Acephate

Aircraft Spill:

Dermal (partial)	5.7×10^{-6}	1.0×10^{-5}
Dermal (full)	1.2×10^{-5}	2.2×10^{-5}
Water (drinking)	3.2×10^{-8}	5.9×10^{-8}

Truck Spill:

Dermal	1.0×10^{-3}	1.9×10^{-3}
Water (drinking)	2.4×10^{-7}	4.4×10^{-7}

DISCUSSION

Threshold Responses

All Exposure Scenarios

Internationally accepted concepts for evaluating exposure, such as the ADI (Acceptable Daily Intake), are applicable for the general human population. As presented earlier in this risk analysis (p. F-67), the ADI is a value that is empirically derived by applying a safety factor (10, 100, 1000, etc.) to a NOEL (no observable effect level), usually derived from a long-term animal feeding study. As such the ADI is based upon a dose that has demonstrated no observable effect in another mammalian species. More importantly, the ADI, through the use of safety factors, makes adjustments for potential variation in response between: 1) the test animal, from which the NOEL is measured, and the human population to which it is applied; and 2) individuals in the human population itself. The adjustment for individual variation in the human population includes that attributable to age and sex, among others. Consequently, doses calculated as equal to or below the established ADI are considered to be within a margin of safety by Regulatory agencies in the United States and worldwide for the general population. It is recognized in this analysis that specific individuals in the population may be more sensitive to xenobiotics (e.g., chemical insecticides) than the general population and that this sensitivity may not be reflected in the ADI. This subgroup will be discussed separately.

For those doses that exceed the ADI, it is necessary to examine the toxicological data base more closely. The relationship of realistic and worst case doses to the lowest NOEL values from animal experiments (Tables 8-11) can be used to judge human safety only when margins of safety (MOS) are related to safety factors traditionally used to establish ADIs. As was discussed on page F-67, these safety factors are as follows: acephate, 10; carbaryl, 100; diflubenzuron, 100; trichlorfon, 100. In other words, if a realistic or worst case dose to

humans is less than the NOEL established for test animals by a MOS of greater than 100, then the dose could be considered to be within a margin of safety for the general population. If the MOS is in the range of 10 to 100, the dose may still probably be within a margin of safety for most people, although there is a greater probability that some individuals in the population could show a response. It is important to examine the particular response for which the NOEL was established in order to discuss the overall impact on human health.

Clearly this risk analysis demonstrates that all of the realistic doses that the general public could receive, exclusive of accidents, are below the established ADI for each insecticide. Project observers and, depending upon the insecticide, mixer/loaders also receive realistic doses that are below the ADI. Only when worst case doses are examined (for all groups) does the analysis identify exposure scenarios that could exceed the ADI. For the general public these exposure scenarios are confined to those that include the dietary component. The mixer/loader group could receive realistic doses of acephate and trichlorfon that are above the ADI. The question that must be answered is whether or not these doses provide a margin of safety. To do this, it is necessary to review the toxicological data base, the data points, and worst case assumptions made to conduct this risk analysis.

One of the most sensitive dose responses for acephate and trichlorfon is for cholinesterase inhibition. This is a low dose response characteristic of organophosphate insecticides. The lowest NOELs, to which the acephate and trichlorfon doses are compared, are for cholinesterase inhibition. The toxicological data for acephate in Table 1 lists cholinesterase inhibition NOELs ranging from 0.25 mg/kg/day in the rat to 0.75 mg/kg/day in the dog. Applying the higher NOEL to the doses in question result in margins of safety that approach the 10 safety factor used by EPA to derive the ADI. For trichlorfon the cholinesterase inhibition NOELs range from 1.0 mg/kg/day in young dogs, to 2.5 mg/kg/day in male rats, a 2.5 times difference (Table 4). Applying the higher NOEL to the doses in question result in margins of

safety that exceed 10. Cholinesterase inhibition is a very readily measurable effect. The NOELs for both insecticides are based upon the first appearance of depression of cholinesterase levels in red blood cells, plasma, and the brain. The NOELs do not represent the appearance of classical clinical symptoms of organophosphate intoxication. Clinical symptoms generally do not manifest themselves until depression exceeds 50 percent.

The cholinesterase inhibition NOEL for both insecticides is at least 10 times lower than the NOEL for more pronounced systemic effects and greater than 100 times lower than the highest doses tested (with no effect) in teratogenicity studies. This suggests that the doses in question are below threshold no effect levels by a margin of safety that is greater than 100 when considering health effects other than the onset of depression of cholinesterase levels. These health effects from exposure to acephate and trichlorfon involve only the worst case daily consumption of meat, vegetables, fruits and liquids containing insecticide residues. This situation has a probability of occurrence of 1.8×10^{-3} or 1 chance in 556.

The doses in question for carbaryl are all very close to the established ADI and more than 10 times less than the lowest NOEL. The lowest NOEL is for teratogenicity in beagle dogs, however it is significant to note that although EPA uses a 100 safety factor to derive the ADI, the agency does not apply that safety factor to the lowest NOEL. The toxicological data in Table 2 identifies five other teratogenic and fetal toxicity NOELs in 4 other animal species besides the dog, all of which are more than 48 times higher than the dog NOEL. Using one of the other teratogenic NOELs for comparison to the doses in question results in margins of safety from 750 to more than 1200. The data base and the fact that EPA does not use the dog study NOEL in order to derive the ADI suggests a lack of confidence in the data. Since the doses in question are very close to the ADI, reexamination of the data base suggests that these doses are within an acceptable margin of safety. This statement is further supported by an epidemiology study conducted in Cape May County, New Jersey, by the New Jersey Department of Health (Halpin

1980). The study concluded that there was no increase in birth defects in the counties where carbaryl was used, compared to counties where it wasn't. Finally, any question of possible health effects to the general public from exposure to carbaryl involves worst case doses only, in addition to the daily consumption of meat, vegetables, fruits, and liquids which contain insecticide residues.

In the case of the general public exposure scenarios, it is the dietary component that drives the worst case dose above the ADI for acephate, carbaryl and trichlorfon. The data points used and the worst case assumptions made result in dietary doses that are conservative at best, and are extreme overestimates at worst. Pesticide residue data gathered by the FDA (Food and Drug Administration) in market basket surveys help to point this out. Carbaryl residues measured in all types of food (the other insecticides in question were lower) represent a daily intake (dose) of 0.00005 mg/kg/day (E. Gunderson, FDA, personal communication) which is 170 times below the lowest dietary dose used in this analysis. After reviewing the toxicological data base, and the data points and worst case assumptions made to carry out this risk analysis it is clear that: (1) all of the doses in the analysis, including those in question, represent overestimates; (2) even the worst case doses in question are probably still within margins of safety discussed; and (3) finally, the probability of the general public and occupationally exposed individuals receiving worst case doses is 1.8×10^{-3} or about 1 worst case dose for every 556 realistic doses received.

Accident-Related Exposures

The most acute impacts to human health are associated with doses that could be received from exposure to insecticide spills. The risk analysis identifies some accident scenarios where exposures and doses could exceed dermal LD_{50}s and ADIs. Some doses are below the lowest NOELs by margins of safety that are much less than the safety factors used to establish the respective ADIs. Doses and exposures associated

with spills of diflubenzuron are below the established ADI and from several to hundreds of times below the highest exposure level tested (with no effect) for the dermal LD_{50}. This indicates that exposure to diflubenzuron spills results in doses that are within margins of safety for the general population.

The dermal exposures that could result from aircraft spills of acephate, carbaryl or trichlorfon are all many times below the highest exposure level tested (with no effect) for dermal LD_{50}s. All doses associated with consumption of water are from slightly above to several times below the established ADIs. Where the water consumption doses exceed the ADI, a similar case to that previously presented can be made, and which suggests that the doses may still be within acceptable margins of safety for the general population. Any further discussion of potential health effects associated with aircraft spills needs to take into account the probability of the accident occurring.

It is not surprising that the largest potential doses and exposures to individuals are associated with tank truck spills. The dermal exposures (realistic and worst case) for acephate, carbaryl and trichlorfon are very close to or exceed the highest doses tested (with no effect) for the dermal LD_{50}. It is not known what effects could result from these exposures, so it is appropriate to identify these exposures as being hazardous. Since acephate, carbaryl, and trichlorfon are listed as moderately toxic cholinesterase-inhibiting pesticides (Morgan 1982), symptoms of acute poisoning could include headache, dizziness, uncoordination, muscle twitching, nausea, abdominal cramps, diarrhea or sweating would develop within 12 hours of exposure. Unconsciousness, convulsions, or respiratory depression could occur if exposure is severe, as is the case with truck accidents. In the case of acephate and trichlorfon peripheral neuropathy could develop following high exposure to these organophosphate insecticides. Morgan (1982) reported that development of this type of neurotoxicity is rare. Doses associated with consumption of water into which spills have occurred

should similarly be considered hazardous. As with aircraft spills, further discussion of potential health effects associated with truck spills needs to take into account the probability of the accident occurring.

The potential hazards associated with exposure to insecticide spills (both aircraft and vehicle) are real and have been identified. Fortunately, the probability of insecticide spills is extremely low. A low probability does not change the hazardous nature of the exposure, but rather estimates the likelihood that exposure to the hazard would occur. This provides the necessary information for readers and decision makers to use in judging the acceptability of these risks. Safety measures can also be taken in the event of accidents which reduce the risk of adverse health effects. Organophosphate and carbamate insecticide poisoning is readily treatable with the antidote, atropine sulfate, in the event of an accident such as a spill or exposure to a worker. Such information should be contained in safety plans for individual suppression or eradication projects.

As demonstrated in this analysis, the accident scenario associated with the greatest hazards to human health (truck spills) has the lowest probability of occurrence. For realistic doses the probability of occurrence is 1.08×10^{-5} on land or 1 spill for every 93,000 vehicle trips, and 1.2×10^{-6} in water or 1 spill for every 833,000 vehicle trips. For worst case doses the probability drops to 1.9×10^{-8} ($1.08 \times 10^{-5} \times 0.0018$, the probability of worst case occurrence) on land, or 1 worst case spill for every 50 million vehicle trips, and 2.2×10^{-9} ($1.2 \times 10^{-6} \times 0.0018$) in water, or 1 worst case spill for every 500 million vehicle trips. The probability of aircraft spills occurring for realistic doses are: 5.1×10^{-4} on land or 1 spill for every 1,960 aircraft loads, and 5.7×10^{-5} in water or 1 spill for every 17,554 aircraft loads. For worst case doses the probability of occurrence drops to 9.1×10^{-7} on land, or about 1 spill for every 1 million aircraft loads, and 1.0×10^{-9} in water, or 1 spill for every 100 million aircraft flights.

Teratogenicity

The issue of teratogenicity, or birth defects, is generally raised by the public when human health effects associated with exposure to chemicals, any chemicals, are being discussed, evaluated, and weighed. The teratogenicity issue generates a great deal of emotion and is therefore appropriate for further discussion here. On pages F-70 to F-73, worst case doses to the highest exposure group, mixers/loaders, were compared to the lowest NOEL found in the literature for teratogenicity (Table 7). Since few women work as mixer/loaders, it is important to compare the doses of a general public group which includes women with the teratogenic NOEL. The general public group with the highest exposure is the observer and dietary group. The margins of safety (MOS) are calculated for each of the insecticides and presented below:

	mixer/loader	observer and dietary	
	worse case	realistic	worst case
acephate	67	1111	76
carbaryl	16	260	18
diflubenzuron	300,000	5,000,000	200,000
trichlorfon	40	666	46

For acephate and diflubenzuron, the margins of safety are well above the safety factors used to establish the ADIs (10 and 100 respectively) for even the highest exposure groups. Neither acephate nor diflubenzuron has caused teratogenicity in laboratory animals. The NOELs listed in Tables 1 and 3 represent the highest doses tested (without an observed effect). Exposures can thus be considered to be within margins of safety for exposure that might result from using these two insecticides to suppress or eradicate gypsy moth.

For carbaryl and trichlorfon the margins of safety for the realistic observer/dietary doses are greater than the safety factors of 100 established for these two insecticides and can therefore be considered to be within acceptable margins of safety. Margins of safety for the worst case mixer/loaders and observer/dietary doses are below 100. However, these low margins of safety may be misleading. The teratogenic NOEL for carbaryl came from the dog, but teratogenic NOELs for other species are 50 to 150 times higher than that of the dog. If the next lowest NOEL (mouse or rabbit = 150 mg/kg/day) were used to compare to the worst case doses, the margins of safety would exceed 800. For trichlorfon, the teratogenic NOEL of 8 listed in Table 7 is actually the highest dose tested for the particular experiment in rats. The study by Staples and Goulding (1979) actually established a teratogenic threshold dose (LOAEL) in rats of 432 mg/kg/day. This suggests that even the worst case doses are within margins of safety for the general population. Therefore it can be concluded that exposures (even worst case) resulting from the use of any of the four insecticides to suppress or eradicate gypsy moth are below teratogenic thresholds and probably within margins of safety for the general population.

Nonthreshold Responses

Risk of Cancer

The assessed risk of cancer that could occur from using carbaryl or trichlorfon to control gypsy moths is only meaningful to the decision maker or other readers if it can be compared to similarly determined risks for known cancer-causing agents (such as X-rays or smoking) or other risks of death. Some risks are so small that people tend to ignore them because they are unconsciously accepted (e.g., crossing a street). For example, many risks of 10^{-6} per year are familiar and casually accepted by the general public.

Weighted lifetime risks of cancer to an individual exposed to acephate, carbaryl, or trichlorfon (Table 16) used in gypsy moth eradication or suppression programs can be compared to a number of risks familiar to society which are listed in Table 17 taken from Crouch and Wilson (1982). The cancer risks shown in Table 17 were calculated by a method similar to that used in this analysis; so the same uncertainties dealing with exposure, measurements of potency, and extrapolation between laboratory animals and humans apply. In all cases, the lifetime risks of cancer resulting from exposure to carbaryl or trichlorfon used to control gypsy moths are lower than the risk of cancer from smoking 2 cigarettes, drinking 40 diet sodas, or having a single X-ray in a lifetime, which are all in the order of 10^{-6}, or one in a million risk.

The individual lifetime risk of cancer resulting from exposure to acephate are slightly greater than the one in a million risks listed above. It is important to point out that even the realistic doses used to calculate the cancer risk would have to be considered worst case. The realistic doses assume that every person in the treatment area will eat food or drink water that has acephate residues that exceed 11 ppm. Lifetime risks to individuals, even those residents who stand outside (observer) during application, who do not consume food or water containing acephate residue would be lower than one in a million by about a factor of 10.

Further, the formulas for calculating total incidences of cancer per project indicate that the cancer risk associated with spraying trichlorfon is less than one case of cancer per million acres treated. The risk associated with spraying with carbaryl is less than one case per 100 million acres treated. The risk associated with spraying acephate is about one case per 500,000 acres treated. Forest Service records show that over the last 4 years, carbaryl has been used yearly on an average of 81,812 acres while trichlorfon has been applied to an average of 160,867 acres and acephate has been used on less than 1,000 acres. Using these acreage numbers, the added risk of cancer from the use of carbaryl would be 0.0004 incidences of cancer ($4.84 \times 10^{-9} \times$

81,812) in the estimated exposed population of 1.14 million people (14 people/acre x 81,812 acres). There would be 0.03 incidences (1.88 x 10^{-7} x 160,867) of cancer in the estimated exposed population of 2.25 million people (14 people/acre x 160,867 acres) living on the 160,867 acres treated with trichlorfon. The added risk of cancer from the use of acephate would be 0.003 incidences of cancer (1,000 acres x 3.2 x 10^{-6} incidences/acre) in the estimated exposed population of 14,000 people. In all cases, the estimated incidences of cancer are based on the assumption that all people living in the treated areas receive the exposure of the highest exposure groups. In actual residue monitoring experiments Schulze (undated) reported detectable carbaryl exposure in 33 percent of the residents and SCESC (1978 and 1979) found that only 20 percent of the residents had detectable exposures. Therefore risk assessments in this analysis overestimate cancer risks.

Accidents.--To fully evaluate the risk of cancer resulting from acephate, carbaryl, or trichlorfon exposure resulting from an accident, the probability of the accident occurring must be considered along with the cancer risk if an individual is exposed. For example, the risk of cancer to an individual dermally exposed to a single large dose of trichlorfon resulting from a truck accident is 1.87 x 10^{-4} (p. F-85). This risk, as well as that associated with truck spills involving acephate, is considerably higher than the lifetime risks of cancer from smoking 2 cigarettes, drinking 40 diet sodas, etc., listed in Table 17. However, the probability of a truck accident occurring is 1.08 x 10^{-5} or about one accident in every 93,000 trips. When the probability of the accident occurring is considered along with the risk of cancer (for example 1.87 x 10^{-4} for trichlorfon), if the resultant exposure occurs, the probable incidence of cancer to an individual resulting from a truck accident because of the use of trichlorfon becomes 2.02 x 10^{-9} (1.08 x 10^{-5} x 1.87 x 10^{-4}).

All other lifetime cancer risks associated with accidents are about equal to 8,000 times lower than familiar risks listed in Table 17 that seem to be accepted by the general public (or a cancer risk of 1 x 10^{-6}

or one in a million). When these cancer risks are considered along with the low probability of an accident occurring, any possible incident of cancer resulting from a possible accident becomes remote.

Risk of Heritable Mutations

Since there is no available epidemiological data demonstrating an association between chemical exposure and heritable mutations, the extent that exposure to natural or synthetic chemicals may increase the number of heritable mutations in the present population is unknown at this time. Therefore, the experimental evidence presented in the Review of Toxicology section indicates only the absence or presence of mutagenic capability, no quantification.

Since diflubenzuron tested negative in all mutagenicity studies, it is considered to be non-mutagenic. No increase in the rate of spontaneous mutations is expected from the use of acephate or carbaryl even though these insecticides are mutagenic in tests with submammals or plants. This conclusion is based on negative mutagenicity results with acephate in whole animals studies. For carbaryl there is insufficient information that the insecticides can reach germinal tissue in humans.

Trichlorfon appears to be a more potent mutagen than the other three insecticides under consideration, testing positive in all tests except the whole animal, mouse micronucleus test, reviewed by Jones et al. (1984) and in bone marrow or spermatogonia in mice reported by Degraeve et al. (1981). There is also suggestive evidence that it can reach germinal tissue. Since mutagenicity and carcinogenicity both follow similar mechanistic steps (at least those that involve genetic toxicity), the increased risk of cancer can be used to approximate the quanitative risk of heritable mutations. The basis for this assumption is that both mutagenicity and at least primary carcinogens react with DNA to form a mutation or DNA lesion affecting a particular gene or set of genes. The genetic lesions then require specific metabolic processes to occur or the cells must divide to the lesion into the genetic code of

the cell. We believe the cancer risk provides a worst case approximation to heritable mutations because cancer involves many types of cells where as heritable mutations involve only germinal (reproductive) cells. Risks of heritable mutation resulting from exposure to trichlorfon used to treat gypsy moth would therefore exceed 1×10^{-7} for each individual.

Sensitive Populations

Thus far all discussions of risk of adverse human health effects have been limited to the general population. The basis for assessment of risk has involved the extrapolation of median effects or no-effects in animals to the human population. Safety factors have been utilized to account for the many biological factors (i.e., young children or elderly adults, sex, genetic composition, and pre-existing disease conditions) that may increase human susceptibility to adverse health effects resulting from exposure to insecticides. Traditionally the intraspecies variation in response to toxic substances has been accounted for by a factor of 10 (Doull et al. 1980 and Dourson and Stara 1983).

There are individuals or groups of people within the general population whose response to exposure for any of the insecticides used in gypsy moth eradication or suppression programs might be greater than that of the general public. In other words, adverse health effects might occur in these sensitive populations at doses much lower than those causing impacts to the general public. Common day examples of this difference in sensitivity within the general population are individuals who are sensitive to pollen, poison oak or ivy, bee stings, or penicillin. Cases of severe hypersensitivity are rare (Ottonboni 1984), and it is impossible to identify a specific percentage of the population that would fall within the sensitive population (Calabrese, 1978).

In order to account for possible impacts to sensitive individuals, the NOELs used in this analysis were reduced by an arbitrary safety factor of 100. ADIs already include a safety factor (100 in most cases) which was incorporated, in part, to account for differential responses within the population. Although the safety factor of 100 is arbitrary, it is based on a review of selected literature on variable human responses to foreign chemicals (xenobiotics) or diseases (NAC-NRC 1977, Glowinski et al. 1978, LaDu and Eckerson 1984, Kersey et al. 1974, Tabershaw and Cooper 1966, and Calabrese 1984). Depending on the specific substance or disease, there was a 3.7- to 100-fold variation in response. However, 80-95 percent of the variation fell within a 10-fold factor.

Examination of the exposure results in Tables 8-10 shows that all realistic doses and those worst case doses that include only dermal exposure (direct, drift and indirect) are below ADIs established by EPA or WHO. Therefore they are considered to be within traditionally accepted safety limits for even sensitive populations. When worst case dietary exposure is considered or added to doses that could result during the direct application of the insecticides (including drift or secondary exposure), worst case doses exceed ADIs and lowered NOELs (reduced 100-fold) indicating that adverse human health effects associated with depressed cholinesterase levels and other systemic toxicity could occur in sensitive individuals. Because of this potential adverse health effect, mitigating measures should be taken so that sensitive individuals, if they can be identified, can avoid direct exposure during application and the eating of food that may contain spray residues. The consumption of food presents a dilemma because food purchased at local retail outlets could contain residues of these same insecticides which are registered for and used on agricultural crops.

Two groups of individuals are presumably at greater risk to diflubenzuron than the general public. There are several genetic defects that predispose to MHb (methemoglobinemia). Most frequently this is caused by a defect in the reductase system, and such individuals may carry a 50 percent MHb burden. Very young infants are also

deficient in MHb reductase, which has resulted in clinical disease in infants given formula made up with water containing excess nitrate (Dr. Frank Dost, Personal Communication).

It is unlikely that gypsy moth suppression and eradiation projects would pose a risk for either group. Persons with genetic tendency to MHb are well aware of their problem. Although the low levels of exposure identified in this analysis should not have in impact, such persons and their physicians should be made aware of the program and an option of removal for several hours during application should be provided. Sensitive individuals should be identified through scoping activities and other forms of public involvement and notification conducted during site-specific environmental analyses, and appropriate measures implemented to mitigate insecticide exposure.

The potential for exposure of infants is very slight, if they are not deliberately carried out into the application. Exposure within a house would be essentially zero. Exposure via water will also be so slight as to represent no hazard.

It is important to note that even for sensitive populations, the risk of birth defects resulting from use of these insecticides is low. The margins of safety between the reduced teratogenicity NOEL (100-fold safety factor applied) and the highest doses (worst case observer and dietary) would be 15 for acephate, 11 for carbaryl, 3703 for diflubenzuron, and 23 for trichlorfon. (These MOSs were calculated on LOAELs as discussed on p. F-93 and 94). The margins of safety for acephate and diflubenzuron are greater than the safety factors used to calculate ADIs. Since the MOS for trichlorfon and carbaryl are less than the safety factor of 100 used to establish ADIs, worst case exposures to sensitive individuals pose some level of teratogenic risk. However, it is important to note that these MOS's are still above the level of 10 which has traditionally been used to account for intraspecies variability (Doull et al. 1980). This margin of safety,

along with the low probability of worst case exposure occuring (2 chances in a thousand), indicate that risk of birth defects in even sensitive individuals is very low. Since realistic doses for this same highest exposure group are about ten times lower than the worst case doses, margins of safety for teratogenicity fall within the range (greater than 100) that has historically been considered to be safe.

Synergism/Cumulative Effects

To complete this analysis, some discussion is needed concerning how these insecticides interact with other chemicals in the environment, or accumulate effects from the same insecticides already in the environment from other sources. Synergism, which concerns many people, is a special type of interaction where the combined effect of a specific insecticide with one or more chemicals in the environment (such as pollutants) would be greater than the sum of the individual effects of the insecticide and chemical(s) (in other words, 2 + 2 is greater than 4). Since we live in a sea of chemicals, the possibility of chemical/insecticide interaction is certainly probable. However, because of the complex number of possible interactions, the result is not readily predictable.

A good measure of whether synergistic or cumulative effects or any total human health affects occur are epidemiological studies on exposed populations. The New Jersey State Department of Health, Parental, and Child Health Services (Halpin 1980) conducted an epidemiology study investigating the relationship between birth defects and carbaryl spraying in areas of New Jersey treated for gypsy moth control. They found no association between spraying of carbaryl and birth defects. Unfortunately, similar studies have not been conducted for the other insecticides or for other possible adverse human health effects. Therefore these possible effects are discussed in a general sense.

Since the dose or concentration of any chemical dictates both the probability and rate of any chemical reaction (and all biological responses in an organism are the result of chemical reactions), the dose of the specific insecticide in the environment or in the individual is an important factor in considering synergistic effects. Ames (1983) pointed out that there are many naturally occurring chemicals in the food people eat which are teratogenic, mutagenic, and carcinogenic and which are consumed at doses 10,000 times higher than man-made pesticides. Therefore, the low, short-lived doses that result from the spraying of these insecticides to control gypsy moth is very small compared to many other chemicals in the environment. For these small comparative doses, a synergistic effect is not realistically expected (Crouch et al. 1983). EPA apparently came to the same conclusion, because they issued a Notice (PR Notice 82-1) on January 12, 1982 (US EPA 1982b), rescinding the requirement for submission of tank mix compatability data. The Notice stated that EPA had examined considerable data and found no evidence of potentiation involving pesticides.

However, Statham and Lech (1975a, 1975b, and 1976) have reported the potentiation or synergism effect of carbaryl on the acute toxicity of 2, 4-D, dieldrin, rotenone, and pentachlorophenal. The acute toxicity of these latter pesticides were increased by factors of 3- to about 8-fold with additions of 1 mg/liter (1 ppm) of carbaryl.

Since the Statham and Lech studies delt with concentrations that are higher than would be expected from the use of carbaryl for gypsy moth, the synergistic effects could be considered to be worst case. However, a synergistic response similar to the interaction of tobacco smoke and asbestos in humans was used for the worst case because Statham and Lech studied effects in fish. Smokers who were also exposed to asbestos have an 8-fold higher risk of lung cancer than smokers alone (Selikoff et al. 1968). If a 10-fold synergistic factor were applied to the NOEL/dose comparisons and cancer risk values developed in this analysis, cancer

risks would still be equal to or less than the one in a million level listed in Table 17. Worst case dose of acephate and trichlorfon that include dietary exposure would exceed the NOEL for cholinesterase inhibition by factors of 2 to 5, indicating that some reduced cholinesterase level effects could occur. All teratogenicity thresholds would still exceed even the worst case dose levels by factors that have traditionally been accepted as safe.

The one resident who reported using carbaryl on his own garden (p. F-30) illustrated just one source of exposure other than that resulting from gypsy moth control. Other major sources of exposure would be through food consumption, or drift for those individuals who live near or adjacent to agricultural areas. To fully discuss cumulative effects, the total dose from all sources would need to be calculated. Since this is impossible, cumulative effects can only be discussed in general terms.

The 1-naphthol residue (2,556 ppb) in the urine of the resident who used carbaryl on his garden shows that doses of 0.01 mg/kg/day are possible from such uses. Food tolerances for residues of acephate (1-10 ppm), carbaryl (0.2 to 12 ppm), diflubenzuron, (0.05 ppm), and trichlorfon (0.1 ppm) show that oral doses could range from 0.007 to 0.07 mg/kg/day (based on the assumptions used in this analysis) if food containing the maximum tolerance was eaten. Such dose levels are equal to or greater than those estimated to result from the use of these insecticides in gypsy moth control programs. This indicates that for acephate, carbaryl, or trichlorfon, it would be possible to accumulate a dose that would exceed the ADI, but not the lowest NOEL or teratogenicity NOEL.

Data gathered by the Food and Drug Administration (E. Gunderson, FDA, personal communication) show that high accumulations of residues of carbaryl, acephate, or trichlorfon have not occurred from eating meats, fruits and vegetables that have been treated with these insecticides.

Residues for carbaryl collected since 1978 project a daily dietary intake of 0.00001 to 0.00005 mg/kg/day. No residues of acephate or trichlorfon have been found in foods that make up the total diet of individuals (these data will soon be published by FDA in J. Assoc. Offical. Anal. Chem.). Since carbaryl is used on more crops than any of the other insecticides, the 0.00005 mg/kg/day residue level can be assumed to be an upper limit of accumulation of these insecticides from agricultural uses. This value is 170 times below the lowest dietary value of 0.0085 mg/kg/day used in this analysis. Therefore, there would be little if any cumulative effect from this source.

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Table 1.--Summary of established no observable effect levels (NOELs) for acephate for selected organisms.

Animal	Type of Study	Effects Studied	NOEL	Reference 1/
Dog	2-year feeding	Inhibition of cholinesterase activity in plasma, red blood cells, and brain	30.0 ppm (0.75 mg/kg/day)	Fed Reg 47(227) 52994-52995, 24 November 1982
Dog	2-year feeding	Systemic toxicity	100 ppm (2.5 mg/kg/day)	Fed Reg 47(227) 52994-52995, 24 November 1982
Rat	28-month feeding/ oncogenic	Inhibition of cholinesterase activity in plasma, red blood cells, and brain	5.0 ppm (0.25 mg/kg/day)	Fed Reg 47(227) 52994-52995, 24 November 1982
Rabbit	Teratogenic	Teratogenicity	10.0 mg/kg (highest dose tested)	Fed Reg 47(227) 52994-52995, 24 November 1982
Rat	Teratogenic	Teratogenicity	200 mg/kg/day (highest dose tested)	Fed Reg 47(227) 52994-52995, 24 November 1982
Rat	Acute delayed neuro toxic	Leg paralysis	375 mg/kg/day (Highest dose tested)	Fed Reg 47(227) 52994-52995, 24 November 1982

1/ See U.S. Environmental Protection Agency (1982a) in References Cited.

Table 2.--Summary of established no observable effect levels (NOELs) for carbaryl for selected organisms.

Animal	Type of Test	NOEL	Reference
Mouse	Teratogenic (birth defects)	5,660 ppm (dietary) 150 mg/kg/day (gavage)	Murray, F.J., Staples, R.E., and Schwetz, B.A. 1979. Teratogenic potential of carbaryl given to rabbits and mice by gavage or by dietary inclusion. Toxico. Appl. Pharmacol. 51:81-89.
Rabbit	Teratogenic (birth defects) (omphalocele)	150 mg/kg/day 200 mg/kg (LOAEL)[1] (maternal wt. loss)	Murray, F.J., Staples, R.E., and Schwetz, B.A. 1979. Teratogenic potential of carbaryl given to rabbits and mice by gavage or by dietary inclusion. Toxico. Appl. Pharmacol. 51:81-89.
Rat	Teratogenic/ Reproductive (maternal weight loss observed) Reproductive (3 generation)	500 mg/kg/day (teratogenic) 10 mg/kg/day (LOAEL) (dilated uterine glands in third generation pups)	Weil, C.S., Woodside, M.D., Carpenter, C.P., and Smyth, H.P., Jr. 1972. Current status of tests of carbaryl for reproductive and teratogenic effect. Toxicol. Appl. Pharmacol. 14:409-419.
Guinea pig	Fetal effects	300 mg/kg/day (Highest dose tested)	Weil, C.S., Woodside, M.D., Bernard, B., Conara, N.L., J.M. King, and Carpenter, C. P. 1973. Comparative effect on carbaryl on rat reproduction and guinea pigs teratology of carbaryl fed either in the diet or by stomach intubation. Toxicol. Appl. Pharmacol. 26:621-638.
Rat	Mutagenic/Teratogenic (3 generation)	200 mg/kg/day (Highest dose tested)	
Guinea pig	Fetal toxicity Maternal toxicity (skeletal defects)	250 mg/kg/day 300 mg/kd/day (LOAEL)	Robens, J.F. 1969. Teratologic studies of carbaryl, diazinon, norea, disulfiram, and thiram in small laboratory animals. Toxicol. Appl. Pharmacol. 15:152-168.

Table 2.--Summary of established no observable effect levels (NOELs) for carbaryl for selected organisms - continued.

Animal	Type of Test	NOEL	Reference
Dog	Teratogenic	3.25 mg/kg/day 6.25 mg/kg/day (LOAEL)	U.S. Environmental Protection Agency 1980 Carbaryl decision document. 66 pp.
Mouse	Dominant lethal*	50 mg/kg/day 1000 mg/kg/day (LOAEL)	Epstein, S.S., E. Arnold, J. Andrea, W. Bass, & Y. Bishop. 1972. Detection of chemical mutagens by the dominant lethal assay in the mouse. Toxicol. Appl. Pharmacol. 23:288-325.

[1] Lowest observable adverse effect level.

* A dominant lethal test only measures a portion of the mutagenic response and cannot be used to establish a NOEL for mutagenicity.

Table 3.--Summary of established no observable effect levels (NOELs) for diflubenzuron for selected organisms.

Animal	Type of Test	NOEL	Reference
Rat	2-year feeding	40 ppm (2 mg/kg/day)*	Mulder, M.S., and Gijswijt, M.J. The laboratory evaluation of two promising new insecticides with interference with cuticle deposition. Pest Sci. 4:745, 1973.
Mouse	80-week feeding	50 ppm (2.5 mg/kg/day)*	Uniroyal 1983. Dimilin-25W Technical Data Sheet. 3/83. 4 p.
	80-week feeding	1.1 mg/kg/day (For methemoglobin and sulfhemoglobin)	USEPA 1984d.
Rat	3 generation	160 ppm (8 mg/kg/day)*	Uniroyal 1983. Dimilin-25W Technical Data Sheet. 3/83. 4 p.
Rat	Teratogenic	4,000 mg/kg/day (highest dose tested)	USEPA 1984g.
Rabbit	Teratogenic	4,000 mg/kg/day (highest dose tested)	USEPA 1984g.
Mouse	Mutagenic	1,500-2,000 mg/kg (Highest dose tested)	MacGregor, J.T., D.H. Gould, Ann D. Mitchell and G.P. Sterling. 1979. Mutagenicity tests of diflubenzuron in the micronucleus test in mice, the L5178Y mouse lymphoma forward mutation assay, and the Ames Salmonella reverse mutation test. Mutation Research 66: 45-53.

* ppm converted to mg/kg/day as per USDA 1984b.

Table 4.--Summary of established no observable effect levels (NOELs) for trichlorfon for selected organisms.

Animal	Type of Test	NOEL (mg/kg/day)	Reference
Sheep	Toxicity	200	Radeloff and Woodward[1/]
Cattle	Toxicity	100	Radeloff and Woodward[1/]
Calves	Toxicity	10	Radeloff and Woodward[1/]
Dog	Decreases acetyl-cholinesterase	500	Marsh, et al.[1/]
Dog	Modification of intestinal fermentic function	100	Gheorghien, 1967[1/]
Young dog	Reduces acetyl-cholinesterase activity	1	Jivogliadova, 1970[1/]
Rat	Reduces citochrom-oxydase activity	57	Jdanovici and Udalov, 1970[1/]
Rat	Modification of vitamin content	30	Nijegoro and Kalinon, 1968[1/]
Rat	Modifies immuno-biological responses	20	Olefir, 1971[1/]
Rat	Teratogenic embryotoxic	8 (Highest dose tested)	Marston, L.V., and V.M. Varonina. 1976. Experimental study of the effect of a series of phosphoroorganic pesticides (Dipterex and Imidan) on embryogenesis. Environ. Health Perspect. 13:121-125.[2]

Table 4.--Summary of established no observable effect levels (NOELs) for trichlorfon for selected organisms - continued.

Animal	Type of Test	NOEL (mg/kg/day)	Reference
Mouse	Mutagenic	19.25 (Highest dose tested)	Becker, J., and J. Schoneich, Zentralinstitut fur Genetik und Kulturpflanzenforschungder AdW der DDR, 4325 Gatersleben (German Democratic Republic). 1980.
Rat (male)	3-month to 2-year feeding study (cholinesterase inhibition)	2.5	Doull, J., Klaasseen, C.D., Amdur MO(Eds): Casarett and Dovll's Toxicology, 2d Ed. New York: MacMillan Publishing Company, Inc. 1980. p. 366.
Dog	3-month to 2-year feeding study (cholinesterase inhibition)	1.25	Doull, J., Klaasseen, C.D., Amdur MO(Eds): Casarett and Dovll's Toxicology, 2d Ed. New York: MacMillan Publishing Company, Inc. 1980. p. 366.
Hamster	Teratogenic (malformations)	200 400 (LOAEL)	Staples, R.E., Goulding, E.H. Dipterex teratogenicity in the rat, hamster, and mouse when given by gavage. Environmental Health Perspective 30:105-113, 1979.
Rat	(malformations)	480 (LOAEL)	
Mouse	(Low fetal weight) (Cleft palate)	400 (LOAEL) 500 (LOAEL)	

1/ Referenced in Zamfir G., Apostol S., Filipuc M: Researches on dipterex in view of establishing the allowable maximum concentration. Environmental Quality and Safety Supplement. Vol. III. Pesticides, pp. 845-849, (ca.) 1975).

2/ Test conducted with Russian manufactured trichlorfon of uncertain purity.

Table 5.--Summary of basic doses by accident scenario and insecticide.

Accident Scenario	Basic Dose (mg/kg/day)			
	Acephate	Carbaryl 1/	Diflubenzuron	Trichlorfon
Aircraft Spill				
Dermal (partial)	53	113	2.1	53
Dermal (full)	110	234	3.2	110
Water (drinking)	0.03	0.07	0.0014	0.03
Truck Spill				
Dermal	9740	25960	389	9740
Water (drinking)	0.227	0.604	0.0091	0.227

1/ In aircraft spills, carbaryl contains 3.2 lbs. active ingredient/gallon.

Table 6.--Probability of accidental tank truck and aircraft spills for a 100,000 acre/year project.

Insecticides	Number of Trips and Flights Required		Aircraft Probabilities		Truck Probabilities	
	Truck	Aircraft 1/	Water	Land	Water	Land
Acephate	25	167	9.5×10^{-3}	8.5×10^{-2}	2.6×10^{-5}	2.7×10^{-4}
Trichlorfon	34	222	1.3×10^{-2}	1.1×10^{-1}	4.1×10^{-5}	3.7×10^{-4}
Carbaryl	13	125	7.1×10^{-3}	6.3×10^{-2}	1.6×10^{-5}	1.4×10^{-4}
Diflubenzuron	50	333	1.9×10^{-2}	1.7×10^{-1}	6.0×10^{-5}	5.4×10^{-4}

1/ Based on an aircraft carrying 300 gallons per load, and the following application rates for insecticides:

Acephate = 0.5 gallons per acre
Trichlorfon = 0.67 gallons per acre
Carbaryl (Sevin 4 oil) = 0.375 gallons per acre
Diflubenzuron = 1.0 gallons per acre

Table 7.--Summary of established exposure thresholds and carcinogenic potency slopes used in the analysis.

Threshold 1/	Acephate	Carbaryl	Diflubenzuron 2/	Trichlorfon
Acute oral LD_{50} mg/kg/day	866-945	512	4,640	144-184
Acute dermal LD_{50} mg/kg/day	10,250	9,580	2,000	2,100
NOEL (low) mg/kg/day	0.25 (cholinesterase inhibition)	3.125 (teratogenic -dogs)	1.1 (methemoglobin and) sulfhemoglobin)	1.0 (cholinesterase inhibition)
(next lowest) mg/kg/day	0.75 (cholinesterase inhibition)	150.0 (teratogenic- mouse, rabbit)	2.0 (systemic effects)	1.25 (cholinesterase inhibition)
(lowest teratogenic)	10.0	3.125	4,000	8.0
ADI (long-term exposure) mg/kg/day	0.025	0.1	0.011	0.01
Carcinogenic potency $(mg/kg/day)^{-1}$	0.025	0.057 (N-nitrosocarbaryl)	None	0.047

1/ Sources for oral and dermal LD_{50}s: acephate (Meister 1983)
trichlorfon (Mobay Chem. Corp. 1981)
carbaryl (Union Carbide 1978)
diflubenzuron (Uniroyal 1983)

2/ The LD_{50}s and NOELs for diflubenzuron, and the dermal LD_{50} for acephate, trichlorfon, and carbaryl represent the highest doses tested without an observed effect. The actual LD_{50}s and NOELs are higher than these numbers.

Table 8.--Relationship of expected doses to established exposure thresholds for acephate.

Exposure Scenario	Realistic			Worst Case		
	Expected Dose (mg/kg/day)	Relationship to Established Threshold* ADI	NOEL	Expected Dose (mg/kg/day)	Relationship to Established Threshold* ADI	NOEL
General Public						
Direct	0.0017	15	147	0.0075	3	33
Drift	0.0011	23	227	0.0049	5	51
Indirect	0.00033	76	758	0.0014	18	179
Direct and Dietary	0.025	= ADI	10	0.110	-4	2
Indirect and Dietary	0.024	= ADI	10	0.108	-4	2
Observer and Dietary	0.026	= ADI	10	0.147	-6	2
Dietary Only	0.024	= ADI	10	0.103	-4	2
Occupational						
Mixers/Loaders	0.035	-1	7	0.15	-6	2
Observers	0.0017	15	147	0.044	-2	6

* Positive numbers indicate the number of times the expected dose is below the established ADI and NOEL. Negative numbers are number of times the expected dose is above established ADI and NOEL.

Table 9.--Relationship of expected doses to established exposure thresholds for carbaryl.

Exposure Scenario	Realistic			Worst Case		
	Expected Dose (mg/kg/day)	Relationship to Established Threshold* ADI	NOEL	Expected Dose (mg/kg/day)	Relationship to Established Threshold* ADI	NOEL
General Public						
Direct	0.0022	46	1420	0.010	10	312
Drift	0.0014	71	2,232	0.0054	19	579
Indirect	0.00044	227	7,102	0.0018	56	1736
Direct and Dietary	0.012	8	260	0.124	-1	25
Indirect and Dietary	0.0098	10	319	0.1178	-1	27
Observer and Dietary	0.012	8	260	0.174	-2	18
Dietary Only	0.0094	11	332	0.116	-1	27
Occupational						
Mixers/Loaders	0.046	2	68	0.20	-2	16
Observers	0.0022	46	1420	0.058	2	54

* Positive numbers indicate the number of times the expected dose is below the established ADI and NOEL. Negative numbers are number of times the expected dose is above established ADI and NOEL.

Table 10.--Relationship of expected doses to established exposure thresholds for diflubenzuron.

Exposure Scenario	Realistic			Worst Case		
	Expected Dose (mg/kg/day)	Relationship to Established Threshold* ADI	NOEL	Expected Dose (mg/kg/day)	Relationship to Established Threshold* ADI	NOEL
General Public						
Direct	0.00013	85	8462	0.0006	18	1833
Drift	0.000087	126	12644	0.00032	34	3438
Indirect	0.000026	423	42308	0.00011	100	10000
Direct and Dietary	0.00072	15	1528	0.0079	= ADI	139
Indirect and Dietary	0.00061	18	1803	0.0074	= ADI	149
Observer and Dietary	0.00072	15	1528	0.0108	= ADI	102
Dietary Only	0.00059	19	1864	0.0073	= ADI	151
Occupational						
Mixers/Loaders	0.0028	4	393	0.012	= ADI	92
Observers	0.00013	85	8462	0.0035	3	314

* Positive numbers indicate the number of times the expected dose is below the established ADI and NOEL. Negative numbers are number of times the expected dose is above established ADI and NOEL.

Table 11.--Relationship of expected doses to established exposure thresholds for trichlorfon.

Exposure Scenario	Realistic			Worst Case		
	Expected Dose (mg/kg/day)	Relationship to Established Threshold* ADI	NOEL	Expected Dose (mg/kg/day)	Relationship to Established Threshold* ADI	NOEL
General Public						
Direct	0.0022	4	454	0.010	= ADI	125
Drift	0.0014	7	714	0.0054	2	185
Indirect	0.00044	23	2,273	0.0018	6	556
Direct and Dietary	0.012	= ADI	83	0.126	-13	8
Indirect and Dietary	0.0098	= ADI	102	0.1178	-12	8
Observer and Dietary	0.012	= ADI	83	0.174	-17	6
Dietary Only	0.0094	= ADI	106	0.116	-12	9
Occupational						
Mixers/Loaders	0.046	-5	22	0.2	-20	5
Observers	0.0022	4	454	0.058	-6	17

* Positive numbers indicate the number of times the expected dose is below the established ADI and NOEL. Negative numbers are number of times the expected dose is above established ADI and NOEL.

Table 12.--Relationship of expected doses to established exposure following possible accidents for acephate.

Accident Scenario	Realistic				Worst Case			
	Expected Exposure (mg/kg/day)	Relationship to Established Threshold 1/			Expected Exposure (mg/kg/day)	Relationship to Established Threshold 1/		
		Dermal LD_{50}	NOEL	ADI		Dermal LD_{50}	NOEL	ADI
Aircraft Spill								
Dermal (Partial)	58	177			106	97		
Dermal (Full)	121	85			220	47		
Water Drinking 2/	0.033		8	=ADI	0.060		4	-2
Truck Spill								
Dermal	10710	1			19480	-2		
Water Drinking 2/	0.246		=NOEL	-10	0.454		-2	-18

1/ Positive numbers indicate the approximate number of times the expected dose is below the established threshold. Negative numbers indicate the approximate number of times the expected dose is above the established threshold.

2/ Since a person may consume contaminated water for more than 1 day, the estimated dose is compared to the lowest NOEL and the ADI and not the acute LD_{50}.

Table 13.--Relationship of expected doses to established exposure following possible accidents for carbaryl.

Accident Scenario	Realistic				Worst Case			
	Expected Exposure (mg/kg/day)	Relationship to Established Threshold 1/			Expected Exposure (mg/kg/day)	Relationship to Established Threshold 1/		
		Dermal LD_{50}	NOEL	ADI		Dermal LD_{50}	NOEL	ADI
Aircraft Spill								
Dermal (Partial)	124	77			226	42		
Dermal (Full)	257	37			468	20		
Water Drinking 2/	0.077		41	=ADI	0.140		22	=ADI
Truck Spill 3/								
Dermal	27000	-3			27000	-3		
Water Drinking 2/	0.604		5	-6	0.604		5	-6

1/ Positive numbers indicate the approximate number of times the expected dose is below the established threshold. Negative numbers indicate the approximate number of times the expected dose is above the established threshold.

2/ Since a person may consume contaminated water for more than 1 day, the estimated dose is compared to the lowest NOEL and ADI, and not to the acute LD_{50}.

3/ These figures deal with spills of insecticide concentrate, therefore only the normal variation factor of 1.04, which represents the manufacture error was used to calculate the expected doses. (see p. F-45 to F-46).

Table 14.--Relationship of expected doses to established exposure following possible accidents for diflubenzuron.

Accident Scenario	Realistic				Worst Case			
	Expected Exposure (mg/kg/day)	Relationship to Established Threshold 1/			Expected Exposure (mg/kg/day)	Relationship to Established Threshold 1/		
		Dermal LD_{50}	NOEL	ADI		Dermal LD_{50}	NOEL	ADI
Aircraft Spill								
Dermal (Partial)	2.3	870			4.2	476		
Dermal (Full)	3.5	571			6.4	312		
Water Drinking 2/	0.0015		733	7	0.0028		393	4
Truck Spill								
Dermal	428	5			778	3		
Water Drinking 2/	0.01		110	=ADI	0.018		61	=ADI

1/ Positive numbers indicate the approximate number of times the expected dose is below the established threshold. Negative numbers indicate the approximate number of times the expected dose is above the established threshold.

2/ Since a person may consume contaminated water for more than 1 day, the estimated dose is compared to the lowest NOEL and the ADI, and not to the acute LD_{50}.

Table 15.--Relationship of expected doses to established exposure following possible accidents for trichlorfon.

Accident Scenario	Realistic				Worst Case			
	Expected Exposure (mg/kg/day)	Relationship to Established Threshold 1/			Expected Exposure (mg/kg/day)	Relationship to Established Threshold 1/		
		Dermal LD_{50}	NOEL	ADI		Dermal LD_{50}	NOEL	ADI
Aircraft Spill								
Dermal (Partial)	58	36			106	20		
Dermal (Full)	121	17			220	10		
Water Drinking 2/	0.033		30	-3	0.06		17	-6
Truck Spill 3/								
Dermal	10130	-5			10130	-5		
Water Drinking 2/	0.227		4	-23	0.227		4	-23

1/ Positive numbers indicate the approximate number of times the expected dose is below the established threshold. Negative numbers indicate the approximate number of times the expected dose is above the established threshold.

2/ Since a person may consume contaminated water for more than 1 day, the estimated dose is compared to the lowest NOEL and the ADI, and not to the acute LD_{50}.

3/ These figures deal with spills of insecticide concentrate therefore only the normal variation factor of 1.04, which represents the manufacture error was used to calculate the expected doses (see p. F-45 and F-46).

Table 16.--Weighted risk of cancer in a 70-year lifetime from exposure to carbaryl or trichlorfon as used in gypsy moth suppression or eradication projects.

Insecticide Exposure Scenario	Weighted Lifetime Cancer Risk	
	Eradication (6 applications)	Suppression (10 applications)
Carbaryl (N-nitrosocarbaryl)		
Dietary	2.07×10^{-9}	3.46×10^{-9}
Trichlorfon		
Direct and Dietary	8.00×10^{-8}	1.34×10^{-7}
Observer and Dietary	8.01×10^{-8}	1.34×10^{-7}
Acephate		
Direct and Dietary	1.4×10^{-6}	2.4×10^{-6}
Observer and Dietary	1.4×10^{-6}	2.4×10^{-6}

Table 17.-- Lifetime risk of death or cancer resulting from everyday activities.

Everyday Risks

Activity	Time to Accumulate a One in a Million Risk of Death	Average Annual Risk per Capita
Living in the United States		
Motor vehicle accident	1.5 days	2×10^{-4}
Falls	6 days	6×10^{-5}
Drowning	10 days	4×10^{-5}
Fires	13 days	3×10^{-5}
Firearms	36 days	1×10^{-5}
Electrocution	2 months	5×10^{-6}
Tornados	20 months	6×10^{-7}
Floods	20 months	6×10^{-7}
Lightning	2 years	5×10^{-7}
Animal bite or sting	4 years	2×10^{-7}
Occupational Risks		
General		
manufacturing	4.5 days	8×10^{-5}
trade	7 days	5×10^{-3}
service and government	3.5 days	1×10^{-4}
transport and public utilities	1 day	4×10^{-4}
agriculture	15 hours	6×10^{-4}
construction	14 hours	6×10^{-4}
mining and quarrying	9 hours	1×10^{-3}
Specific		
coal mining (accidents)	14 hours	6×10^{-4}
police duty	1.5 days	2×10^{-4}
railroad employment	1.5 days	2×10^{-4}
fire fighting	11 hours	8×10^{-4}

Table 17.-- Lifetime risk of death or cancer resulting from everyday activities - continued.

Other One in a Million Risks

Source of Risk	Type and Amount of Exposure: Examples
Cosmic Rays	one transcontinental round trip by air living 1.5 months in Colorado compared to New York camping at 15,000 feet over 6 days compared to sea level
Other	20 days of sea level natural background radiation 2.5 months in masonry rather than wood building 1/7 of a chest X-ray using modern equipment
Eating and drinking	40 diet sodas (saccharin) 6 pounds of peanut butter (aflatoxin) 180 pints of milk (aflatoxin) 200 gallons of drinking water from Miami or New Orleans 90 pounds of broiled steak (cancer risk only)
Smoking	2 cigarettes

From Crouch and Wilson (1982).

Table 18.--A summary of the possible roles for selected short-term tests in chemical hazard assessment.

General Assay Type	Roles	
	ICP	IMP
Microbial Assays		
Ames Reverse Mutation Test	++	+
Reverse Mutation in *E. coli* WP_2 and Related Strains	+	+
Bacterial DNA Repair Tests	+	NA
Yeast Mutation Tests	+	++
Yeast Mitotic Recombination	+	NA
In Vitro Mammalian Cell Assays		
Mouse Lymphoma Assay (TK)	+	++
CHO or V79 Mutation Assays (HGPRT)	+	++
Unscheduled DNA Synthesis (UDS)	++	NA
Chromosome Aberrations	+	++
Sister Chromatid Exchange (SCE)	++	NA
Cell Transformation	++	NA
In Vivo Mammalian Assays		
SCE	+	NA
Dominant Lethal Assay	NA	++
Cytogenetic Analysis (aberrations)	+	++
Micronucleus Assay	+	+
Spermhead Abnormality Assay	NA	(+)
Heritable Translocation Assay in Mice	NA	+
Specific Locus Assay in Mice	NA	++
DNA Adduct Formation	+	(+)
UDS Assays	+	(+)
In Vivo Submammalian Assays		
Drosophila Assays	+	++
Plant Cytogenetics	NA	(+)

ICP = Identifies mammalian carcinogenic potential.
IMP = Identifies mammalian mutagenic potential.
\+ = Applicable.
++ = Greater applicability for this role.
NA = Not applicable.
(+) = Possible application under limited conditions.

(Source: Dr. David J. Brusick, Litton Bionetics, Inc.)

APPENDIX G

COMMENT LETTERS AND RESPONSES TO THE DRAFT SUPPLEMENT TO THE FEIS*

* Comment letters and responses to the 1984 draft EIS are contained in the 1984 FEIS and are therefore not reproduced here. Copies of the 1984 FEIS may be obtained by writing to the USDA Forest Service or APHIS address listed on the cover page of this document.

MARYLAND
DEPARTMENT OF STATE PLANNING
301 W. PRESTON STREET
BALTIMORE, MARYLAND 21201-2365

HARRY HUGHES
GOVERNOR

CONSTANCE LIEDER
SECRETARY

December 19, 1984

Mr. David E. Ketcham
U.S. Department of Agriculture
12th & Independence, SW, Box 2417
Washington, D.C. 20013

SUBJECT: REVIEW OF DRAFT SUPPLEMENT

State Identification Number: MD84-1-262

Applicant: U.S. Department of Agriculture

Description: DEIS - Gypsy Moth Suppression and Eradication Projects

Dear Mr. Ketcham:

Thank you for providing copies of the draft supplement, including worst case analyses, for the referenced subject.

We are forwarding copies of your draft supplement, including worst case analyses, to appropriate agencies inviting them to contact Mr. Gary E. Moorehead (USDA, Animal and Plant Health Inspection Service) directly with any comments or concerns by February 4, 1985. We are also requesting that a copy of any response be forwarded to the State Clearinghouse. This letter will evidence compliance with intergovernmental review procedures. Please use the State Identification Number referenced above in future correspondence.

Thank you for your cooperation with the intergovernmental review process.

Sincerely,

Guy W. Hager
Director, Maryland State Clearinghouse
for Intergovernmental Assistance

GWHwsr

Review Coordinators

Bruce Gilmore - DNR
Wayne Cawley - DOA
William Smith - DSP

Information Copy

Gary E. Moorehead - USDA, APHIS

TELEPHONE: 301-383-7875
OFFICE OF STATE CLEARINGHOUSE

1-2-85

FM206 12/21/84

NORTH CAROLINA STATE CLEARINGHOUSE
DEPARTMENT OF ADMINISTRATION
116 WEST JONES STREET
RALEIGH NORTH CAROLINA 27611

2

ACKNOWLEDGEMENT OF RECEIPT

MAILED TO

US DEPT. OF AGRICULTURE, FOREST SERV.
GARY E MOOREHEAD
FEDERAL BUILDING, ROOM 663 - A
HYATTSVILLE, MARYLAND 20782

FROM

MRS. MARY WATKINS
ADMINISTRATIVE ASSISTANT

PROJECT DESCRIPTION

THIS EIS COVER FOREST SERVICE AND ANIMAL AND PLANT HEALTH INSECTION SERVICE GYPSY MOTH SUPPRESSION AND ERADICATION PROJECTS IN THE U.S.

TYPE - DRAFT SUPPLEMENT TO THE FINAL EIS

THE N.C. STATE CLEARINGHOUSE HAS RECEIVED THE ABOVE PROJECT FOR INTERGOVERNMENTAL REVIEW. THIS PROJECT HAS BEEN ASSIGNED STATE APPLICATION NUMBER 85E00000450. PLEASE USE THIS NUMBER WITH ALL INQUIRIES OR CORRESPONDENCE WITH THIS OFFICE.

REVIEW OF THIS PROJECT SHOULD BE COMPLETED ON OR BEFORE 01/21/85.

SHOULD YOU HAVE ANY QUESTIONS PLEASE CALL (919) 733-4131.

Fwd

3

OREGON PROJECT REVIEW ACKNOWLEDGMENT

State Clearinghouse
Intergovernmental Relations Division
155 Cottage Street N. E.
Salem, Oregon 97310

received 1/4

Statewide

Phone (503)378-3732 or Toll Free in Oregon 1-800-422-3600

Applicant: USDA Forest Service
Project Title: Gypsy Moth Suppression Draft Supplement to Final EIS
Date Received: 12/28/84 (Start of-45 day review period)
PNRS #: OR841228-042-5 BE SURE TO PLACE THIS NUMBER ON YOUR APPLICATION BEFORE SUBMITTING TO FEDERAL AGENCY.

Your project notice has been assigned the file title and number that appear above. Please use it in correspondence and,if applicable, enter it in Block 3A on the 424 form for the project. Your project notice must also be submitted for review to any affected areawide clearinghouse.

[ ] FEDERAL GRANT [ ] HUD HOUSING [X] DIRECT FEDERAL DEVELOPMENT
[ ] ENVIRONMENTAL ASSESSMENT [ ] DRAFT EIS [ ] FINAL EIS
[ ] STATE PLAN/AMENDMENT

ECONOMIC DEV. & CONSUMER SVCS
[X] Agriculture
[X] Soil and Water
[ ] Economic Development
[ ] Fire Marshal
[ ] Housing
[ ] Labor
[ ] Real Estate

EDUCATION
[ ] Education
[ ] Educ. Coord. Comm.
[ ] Higher Education

EXECUTIVE
[ ] Budget

HUMAN RESOURCES
[ ] Adult & Family Services
[ ] Children's Services
[ ] Community Services
[ ] Corrections
[ ] Employment
[ ] Health
[ ] Mental Health
[ ] Senior Services
[ ] Vocational Rehabilitation

NATURAL RESOURCES
[X] Governor's Office
[X] DEQ
[ ] Energy
[X] Fish and Wildlife
[X] Forestry
[ ] Geology
[ ] Lands
[X] LCDC
[ ] Water Resources

TRANSPORTATION
[ ] Aeronautics
[ ] Director
[ ] Highway Division
[X] Historic Preservation
[ ] Parks Division
[ ] Public Transit

MISCELLANEOUS
[ ] Dev. Disabilities Council
[ ] Extension Service
[ ] Other

NOTE: Your project was circulated to state agencies checked above.

State Clearinghouse use only:
State Agency Due Date: 2-1-85
Federal Agency: USDA
County: S/W

...DE

...100

...MENT
MANAGEMENT

Fwd

RCVD. FOSS

received
1/7

4

STATE CLEARINGHOUSE

30 EAST BROAD STREET • • COLUMBUS, OHIO 43215 • 614 / 466-7461

December 28, 1984

David E. Ketcham, Director of
Envrionmental Coordination
U. S. Department of Agriculture
12th and Independence S.W.,PO Box 2417
Washington, D.C. 20013

Attention: R. Max Peterson, Chief of U.S. Forest Service

RE: Review of Environmental Impact Statement/Assessment Report
Title: Draft Supplement to the FEIS of Gypsy Moth Suppression and Eradication Projects
SAI Number: 36-445-0011

Dear Applicant:

Your Environmental Impact Statement/Assessment has been received in the Ohio State Clearinghouse and the review process has now started. You may expect notification no later than 40 days following the receipt date of a draft Environmental Impact Statement/Assessment and 32 days for a final Environmental Impact Statement/Assessment that the review has been completed.

A State Application Identifier Number (SAI) has been assigned to your Environmental Report. Please refer to this number in all future contacts with the Ohio State Clearinghouse.

Sincerely,

Linda E. Wise

Review Coordinator

0160L

TENNESSEE HISTORICAL COMMISSION
DEPARTMENT OF CONSERVATION
701 BROADWAY
NASHVILLE, TENNESSEE 37203
615/742-6716

January 4, 1985

David E. Ketcham
Director of Environmental Coordination
United States Department of Agriculture
Firest Service
12th. and Independence, SW, P. O. Box 2417
Washington, D. C. 20013

Re: Gypsy Moth Supression and Eradication Projects, Draft Supplement to the Final Environmental Impact Statement, Statewide, CH# 85-0924

Dear Mr. Ketcham:

The above proposed undertaking has been reviewed with regard to National Historic Preservation Act compliance by the participating federal agency or its designated representative. Procedures for implementing Section 106 of the Act are codified at 36 CFR 800 (44 FR 6068-6081, Jan. 30, 1979).

Based on the information available, it is our opinion that due to the location, scope, and nature of the undertaking the project will have no effect on National Register or eligible properties. Therefore, unless project plans are changed or National Register eligible properties are discovered during project implementation, no additional action is necessary to comply with the Act.

The applicant or federal agency should keep this letter as evidence of compliance with Section 106. Any questions or comments should be directed to Joe Garrison Your cooperation is appreciated.

Sincerely,

Herbert L. Harper,
Executive Director and
Deputy State Historic
Preservation Officer

HLH:jk

Office for Planning and Programming

Capitol Annex, Des Moines, Iowa 50319 Telephone (515) 281-3711

TERRY E. BRANSTAD
Governor

EDWARD J. STANEK, PhD
Director

January 4, 1985

David E. Ketcham
Director of Environmental Coordination
USDA/Forest Service
12th and Independence S.W.
Washington, D.C. 20013

RE: IA851226-135

Dear Mr. Ketcham:

The State Clearinghouse has completed the review of the draft supplement to the Final Environmental Impact Statement for Gypsy Moth Suppression and Eradication Project. Agencies and individuals that may have an interest in it have had the opportunity to examine and comment upon its contents. As no objections, recommendations or statements of support were received concerning the information contained in it were received, the Clearinghouse has completed its review and has no comments concerning the draft supplement to the Environmental Impact Statement.

A copy of this letter should accompany the document when it is forwarded to the federal agency as evidence that the State of Iowa has had the opportunity to examine it.

Sincerely,

A. Thomas Wallace

A. Thomas Wallace
Federal Funds Coordinator

ATW/cn

JAN 1 · 1985

STATE OF RHODE ISLAND AND PROVIDENCE PLANTATIONS

7

Department of Administration
STATEWIDE PLANNING PROGRAM
265 Melrose Street
Providence, Rhode Island 02907

Director
Programs
Methods
~~Coordination~~
Pesticides
R-9 Pest Coord.
Clerical
MFO
SPFO
DFO
File

January 11, 1985

Mr. Thomas N. Schenarts
Area Director
USDA Forest Service
370 Reed Road
Broomall, PA 19008

Dear Mr. Schenarts:

The R.I. Office of State Planning, the Single Point of Contact, has conducted an intergovernmental review as prescribed by Presidential Executive Order No. 12372 and has the following comment.

We support the selected alternative cited in the Final EIS for gypsy moth suppression which was approved on 8 May 1984. The selected alternative of Integrated Pest Management affords the greatest latitude for effectively controlling the gypsy moth.

The Draft Supplement to the Final EIS provides updated and more useful information particularly on the potential environmental consequences of utilizing chemical insecticides. The analysis of chemical insecticides appeared to be quite thorough and should be helpful to public based information and educational programs. The new information given in this document allows decision makers to establish gypsy moth control programs based on the best available information.

If you have any questions, please feel free to contact me at (401) 277-2656.

Yours truly,

Michael T. Marfeo
Review Coordinator

MTM/sjc
cc:
Robert L. Williamson, Director
National Program Planning Staff
USDA Animal and Plant Health Inspection Service
Federal Building
Hyattsville, MD 20782

John Ashcroft
Governor

RCVD. FOSS 1-24-85

8

State of Missouri
OFFICE OF ADMINISTRATION
Post Office Box 809
Jefferson City
65102

John A. Pelzer
Commissioner

Perry M. McGinnis, Director
Division of Budget and Planning

January 21, 1985

Mr. Gary E. Moorehead
Staff Officer
USDA - APHIS - PPQ
Federal Building, Room 663
Hyattsville, Maryland 20782

Dear Mr. Moorehead:

Subject: 84120037 - Draft Supplement to FEIS - Gypsy Moth Suppression and Eradication Projects

The Missouri Federal Assistance Clearinghouse, in cooperation with state and local agencies interested or possibly affected, has completed the review on the above project application.

None of the agencies involved in the review had comments or recommendations to offer at this time. This concludes the Clearinghouse's review.

A copy of this letter is to be attached to the application as evidence of compliance with the State Clearinghouse requirements.

Sincerely,

Lois Pohl

Lois Pohl, Coordinator
Missouri Clearinghouse

LP:cm

January 28, 1985

Mr. Gary Moorehead
USDA - APHIS -PPQ
Federal Building, Room 663
Hyattsville, Maryland 20782

Dear Mr. Moorehead:

Thank you for the opportunity to comment on the draft supplemental environmental impact statement for the Gypsy Moth Suppression and Eradication Projects. We reviewed the EIS and find that the Department of Ecology has no comments to offer. However, we did coordinate the review of this EIS with other state agencies and received one response from the Department of Agriculture. The Department of Agriculture's letter is attached for your information.

If you have any questions, please call the Department of Agriculture at (206) 753-5063 or me at (206) 459-6237.

Sincerely,

Greg Sorlie

Greg Sorlie, Supervisor
Environmental Review and
Permit Management Section

GS:pk

cc: Dr. Judith Freeman, Agriculture

Booth Gardner
Governor

C. Alan Pettibone
Director

OLYMPIA, WA 98504

STATE OF WASHINGTON
DEPARTMENT OF AGRICULTURE
406 General Administration Bldg. AX-41 • Olympia, Washington 98504 • (206) 753-5063

9A

January 23, 1985

Barbara Ritchie, NEPA Coordinator
Department of Ecology, PV-11
Olympia, Washington 98504

Dear Ms. Ritchie:

We have reviewed the USDA, Forest Service, APHIS Draft Supplement to the Gypsy Moth Suppression and Eradication Projects Final Environmental Impact Statement and would like to suggest the following corrections:

1) Appendix E, page E5, lines 3 and 5 should read:

Pierce County, WA (Tacoma East)
Pierce County, WA (Tacoma West)

2) Appendix E, page E6, line 17 should read:

Snohomish County, WA...B.t. (3)

Our own gypsy moth eradication projects continue to be compatible with the FEIS and Draft Supplement, utilizing what is essentially the integrated pest management option. Our 1984 program, consisting of aerial applications of Bacillus thuringiensis (3 applications per treatment area at 16 BIU's/acre) augmented in some areas by limited spot ground Orthene treatments, met with considerable success. Four of the five areas treated showed sufficient reduction (98% or more) in populations of gypsy moths that the 1985 eradication program in those areas will consist solely of pheromone trapping.

The WSDA continues to believe that a comprehensive detection program coupled with spot eradication programs implementing sound integrated pest management principles can and will protect the state of Washington from the permanent establishment of the gypsy moth.

Sincerely,

CHEMICAL AND PLANT DIVISION

JUDITH FREEMAN, Ph.D.
Entomologist, Special Projects

JF/ph

cc: Art Losey
Robert Rebhan

OREGON INTERGOVERNMENTAL PROJECT REVIEW

State Clearinghouse
Intergovernmental Relations Division
155 Cottage Street N. E.
Salem, Oregon 97310

Phone (503)378-3732 or Toll Free in Oregon 1-800-422-3600

C O N C L U S I O N S

APPLICANT: USDA FOREST SERVICE

PROJECT TITLE: GYPSY MOTH SUPPRESSION DRAFT SUPPLEMENTAL TO FINAL EIS

DATE: February 5, 1985

The State of Oregon (and local clearinghouses if listed) has reviewed your project and reached the following conclusions:

☐ No significant conflict with the plans, policies or programs of state or local government have been identified.

☒ Relevant comments of state agencies and/or local governments are attached and should be considered in the final design of your proposal.

☐ Potential conflicts with the plans and programs of state and/or local government:

- ☐ may exist.
- ☐ have been identified and remain unresolved. The final proposal has been reviewed and the final comments and recommendations are attached.
- ☐ have been satisfactorily resolved. No significant issues remain.

A copy of this notification and attachments, if any, must accompany your application to the federal agency.

FEDERAL CATALOG #_____.

NOTICE TO FEDERAL AGENCY

THE FOLLOWING IS THE OFFICIALLY ASSIGNED STATE IDENTIFIER NUMBER:

OR841228-042-5

IPR #3

Dolores Streeter
Clearinghouse Coordinator

OREGON INTERGOVERNMENTAL PROJECT REVIEW

State Clearinghouse
Intergovernmental Relations Division
155 Cottage Street N. E.
Salem, Oregon 97310

Phone (503)378-3732 or Toll Free in Oregon 1-800-422-3600

STATE AGENCY REVIEW

Project Number: OR 841226-042-5 Return Date: 2/1/85

ENVIRONMENTAL IMPACT REVIEW PROCEDURES

If you cannot respond by the above return date, please call to arrange an extension at least one week prior to the return date.

ENVIRONMENTAL IMPACT REVIEW
DRAFT STATEMENT

( ) This project has no significant environmental impact.

( ) The environmental impact is adequately described.

(X) We suggest that the following points be considered in the preparation of a Final Environmental Impact Statement.

( ) No comment.

Remarks

SEE ATTACHED COMMENTS.

Agency [illegible] By L. Ray N K Leu Director for Insect & Disease Mgmt

IPR #5

The following points might improve the overall consistency and quality of the EIS on Gypsy Moth suppression and eradication projects.

1. A decision should be made on whether information of questionable scientific quality needs to be mentioned in an EIS. The review of toxicological studies (p. F-3 in the supplement) is restricted to refereed journals. However nonscientific observations of possible adverse effects are also included (p. 24 supplement). Information on pesticide effects should be of comparable quality or it will be misinterpreted as conflicting evidence with similar scientific credibility. a

2. A discussion of NOEL or API levels of B.t. should be included in this document. If this information is unnecessary, a clear explanation to this effect should be included in the EIS. b

3. Such terms as NOEL, ADI and LD50 should be fully defined in a footnote whenever they appear in tables. c

National Network To Prevent Birth Defects

11

Box 15309, Southeast Station, Washington, D.C. 20003, 202 543-5450

February 2, 1985

Mr. Gary E. Moorehead
Staff Officer
USDA-APHIS-PPQ
Federal Building, Room 663
Hyattsville, Maryland 20782

Dear Mr. Moorehead,

We are an organization mailing to 2,000 groups and individuals nationally aimed at reducing the impact of hazardous substances, including pesticides, upon rates of birth defects and childhood injuries. With my past eight years of work at Friends of the Earth, I have become well aware of the profound injuries that can be produced by some of the federal spray programs, including gypsy moth.

I have just finished trying to read your latest publication, USDA Gypsy Moth Suppression and Eradication Projects, Draft Supplement to the Final Environmental Impact Statement. Quite frankly, it is written in such a way that it is "gibberish" and not even an expert could understand what you have said. On one hand, you have denied that the EIS statement of the past on the program has been discarded and is no longer applicable, yet this document refers extensively to that past document and makes alterations to the text of that document without providing the reader a copy of that document. a

You know as well as I do that such a writing style is blatently "illegal" and a violation of the regulations of the CEQ that these documents be readable and intelligible. b

Since you have referred to the Environmental Impact Statement without providing the reader any summary of its general findings, this precludes adequate citizen participation. c

When I was at Friends of the Earth, I petitioned the Environmental Protection Agency for a more complete scientific review of the pesticide "carbaryl" which is used extensively in gypsy moth programs. EPA agreed with us, and has requested the manufacturer for more tests on birth defects and kidney damage, particularly. This certainly indicates problems with this chemical. Dimilin is known to cause blood disease in animals-possibly leukemia, and we know that the organophosphate chemicals like Dylox have some severe health effects. Your document clearly does not refect the health risks associated with the use of chemical poisons for gypsy moth suppression. d

Furthermore, we know that a full range of biological and integrated pest management controls are available for the gypsy moth, which will in fact reduce costs of the programs in the long-

run. Indeed, in the past when I was at Friends of the Earth, I wrote David Stockman at OMB about the wasteful public spray programs of the Department of Agriculture. The gypsy moth program seems like all the others, designed to kill natural predators of the pest so that it will spread faster to other territories, and permit USDA to justify an ever increasing budget at the expense of the taxpayer and the expensive of the health of America's families and children. e

Your draft document hardly discusses alternative biological controls, which indeed offer a complete and safe alternative to the rather risky and dangerous program upon which USDA has been determined to embark for so many years. As you know, CEQ regulations require consideration of alteratives. f

In limiting comments to the Draft Supplement, USDA has blocked our right to comment on the complete Environmental Impact Statement, and created a gibberish presentation that would make determination of your conclusions impossible. You claim that you have withdrawn the final EIS due to serious flaws in its analysis of the human health risks, and yet the Draft Supplement refers to that document repeatedly. g

Quite clearly, public comment on a matter of such exposure of the public and such government waste of funds in pursuance of the worst alternative for the gypsy moth program, requires a public hearing for adequate public input. This has not been allowed for. h

Exposure analysis in the Draft Supplement, to the extent that we can understand it from your presentation, appears to suggest that every route of exposure should be considered separately- that foliage residues, water, skin, etc. are all independent of each other, and that only one chemical exposure at a time can be considered. This certainly must be aimed at understating the real risk to people - as seems to be an intent of your past documents on the subject. i

Finally, it is quite clear that the registration program at E.P.A. has been plagued in the past with missing data, fraudulent testing, and inadequate analysis. Many of the chemicals have been tested with less than modern techniques. In the case of carbaryl, of course, we know that not only are serious data gaps admitted by E.P.A. (birth defects and kidney and other organ damage) but also that the agency did not address other data gaps (immune system suppression, cancers due to nitro combinations). Furthermore, the existing data shows clearly that low birth weights and abortions occurred in monkeys, and serious reproductive damage in other animals, a problem compounded by reports of human abortions and birth defects after gypsy moth sprays. It is quite clear that USDA cannot rely upon registration as proof of safety, and indeed Dr. John Moore has pointed out in the past that he has never maintained that registration means that pesticides are "safe". j

Quite apart from the fact that I have long characterized many of the USDA pest suppression programs as involving reckless endangerment to America's families and children, and as involving as large a waste of the taxpayer's money as any agency of the government, this letter points out that your present environmental impact statement program is quite "illegal" - failing to meet the requirements of CEQ and failing to make it possible and feasible for there to be adequate public input as required by CEQ and the law. k

We request a complete rewrite of the EIS statement on the gypsy moth suppression program to adequately reflect public health, the environment, and the less expensive alternatives. Secondly, we request a full public hearing on the program. l

Sincerely,

Erik Jansson

Erik Jansson

copies

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NORTHWEST COALITION for ALTERNATIVES to PESTICIDES
P.O. BOX 375 EUGENE, , OREGON 97440
(503) 344-5044

Gary E. Moorehead, Staff Officer
USDA - APHIS - PPQ
Federal Building, Room 663
Hyattsville, Maryland 20782

February 2, 1985

Comments on the Draft Supplement to the Final Environmental Impact Statement: Gypsy Moth Suppression and Eradication Projects.

I. The Worst-Case Analysis Was Undertaken to Address OEC v. Kunzman, Not NEPA

This Draft Supplement is an exercise in letting decisionmakers think that their decision to use a chemical insecticide against the gypsy moth should not be hampered by concerns over public health.

It will fail, perhaps in the courts, perhaps in the public domain, because it does not take seriously the concerns that are scientifically credible and that won't go away, either by wishful thinking or mathematical chicanery. The risk analyst has decided to address concerns raised in OEC v. Kunzman (F-3), rather than NEPA. The analysis is complex, but NOT SIMPLE ENOUGH: it doesn't present decisionmakers with the various drawbacks of each pesticide (including B.t.) so that a decisionmaker can say about each pesticide, "This pesticide has these efficacy benefits, these environmental drawbacks, and these human health drawbacks," (the latter being the focus of the worst case analysis). Only with these types of information can a decisionmaker balance the pros and cons of various pest management tools (including B.t.) against each other and finally make decisions in an admittedly imperfect world. The intent of Congress, of the courts, and of CFR 1502.22 is to facilitate such decisionmaking.

The worst case analysis should first discuss the various scientifically credible risks that have been identified with each of these pesticides and only later discuss the probability of their occurrence. Decisionmakers should end up with a sense of the various risks that cling to each of these pesticides so that they can prepare to reasonably defend their spray programs, if, after knowing the risks, they decide that spray programs are what are called for. This worst case analysis fails to so prepare them. Pesticides, while sometimes necessary, are hazardous. To deny this is wishful thinking.

NCAP is concerned about evidence that diflubenzuron causes methemoglobinemia and sulfhemoglobinemia in laboratory animals; that diflubenzuron is metabolized into 4-chloroaniline (related to known human bladder carcinogens); that acephate is metabolized into the more toxic methamidophos; that carbaryl has been shown to potentiate the toxicity of other insecticides; that diflubenzuron has been shown to persist for months on and in plants (food plants?), and to bioconcentrate in fish, and that acephate and methamidophos can persist for months in water (well water?). These (and other) concerns are not mentioned in this so-called "worst case" analysis. The only time the word "severe" is used (p. 24), indeed the only time individual human case reports are referred to, is in relation to B.t.

II. The Analysis of Exposures and Risk Arrives at Meaningless Numbers

In the near total absence of data about the fate of any of these five insecticides (i.e. including B.t.) in the environment and about human exposure to these (or other) insecticides in aerial spray operations, the risk analysis nevertheless arrives at single numbers for total human exposure to various types of people (e.g. residents, observers, mixers-loaders). Although they are nearly meaningless, these numbers are then compared to numbers that have been derived elsewhere for Acceptable Daily Intake (ADI) and No Observable Effect Levels (NOEL). Then judgment is passed on whether these insecticides pose an "acceptable risk" for nongenetic effects.

By following the example of acephate, perhaps the charge that the numbers are meaningless can be illustrated. First I will discuss how the risk analysis wrongly dismisses concern for genetic effects of acephate, then I will follow the data-less determination of human exposure to acephate for one category of people (i.e. Worst Case - Direct and Dietary: 0.093 mg/kg/day) using every worst case assumption made by the risk analyst, and finally I will discuss the twisted comparison made of this number to acephate's ADI and NOEL. A similar presentation could be made for three of the other insecticides, but the risk analysis does not provide Bt. with such a "full" treatment.

1. Acephate has, as noted in the risk analysis (F-8), been found to be mutagenic in S. typhimurium and Escherichia coli (reverse mutations) and in Saccharomyces cerevisiae (reverse mutation, enhanced mitotic recombination, and gene conversion and crossing-over). It has been found positive in DNA repair assays in yeast and "mammalian cells in culture without metabolic activation," (i.e. human lung fibroblasts), and to increase sister chromatid exchanges in Chinese hamster ovary cells.

The risk analysis decides to not consider genetic toxicity to be a risk with acephate, however, because it has not yet been shown to be positive in tests in whole mammals (e.g. SCE and chromosome aberration tests in mice and monkeys and a dominant lethal test in mice). The analyst states, "...studies in whole mammals indicate that these effects [i.e. the positive results in eight genotoxicity bioassays]...are not produced at detectable levels in intact mammalian systems." (F-8).

As a result of this reasoning, the risk analysis makes the following conclusion on F-77: "No increase in the rate of spontaneous mutations is expected from the use of acephate or carbaryl even though these insecticides are mutagenic in tests with submammals or plants. This conclusion is based on negative mutagenicity results with acephate in human cells in culture, [this is inaccurate - acephate has been shown to cause unscheduled DNA synthesis in human lung firbroblasts], and whole animals studies." d

This conclusion is not valid and the risk analysis must be modified to consider genetic risk of acephate. The dominant lethal test in mice is one of the most insensitive of all in vivo tests. The SCE test has a spotty record and geneticists don't know how to interpret the meaning of results. The only other whole mammal test cited is the chromosome aberration test and the group that did this test (Jones et al. 1984, cited in the EIS) is not regarded with universal high regard among geneticists. The test would need to be studied for the doses, statistical treatment, etc. It is simply not acceptable to dismiss results of eight postive in vitro tests and claim that acephate poses no genetic risk. That risk may not be quantifiable, but it cannot be dismissed.

The EIS goes on to say (F-9) that acephate will not be considered for cancer risk: "It was concluded from reviewing the data available that acephate (Seiler 1977)...[is] not carcinogenic and therefore do[es] not pose a cancer risk." The Seiler reference is not a cancer bioassay: "Seiler, J.P. 1977. Nitrosation in vitro and in vivo by sodium nitrite and mutagenicity of nitrogenous pesticides. Mutation Research. 48: 225-236." If a pesticide is found to be positive in several in vitro tests, cancer bioassays are needed. If they have not been done, the assumption that acephate may cause cancer is unavoidable. e

2. As for nongenetic effects, acephate is conditionally registered. No-one knows what legally-mandated toxicological studies are missing from acephate's files because a registration standard has not yet been issued by the EPA. In addition, the conditional registration of acephate was based, in part, on 19 studies by IBT, 12 of which were ruled invalid by EPA. Therefore, the No Observable Effect Level (NOEL) to which the final exposure numbers for various scenarios is compared, is tentative at best. This issue is not mentioned.

If the determination of human exposure to acephate via the Worst Case - Direct and Dietary scenario is followed, it is discovered that for the whole scenario, not one piece of data on acephate, from either experimental or field studies, is used except for the rate of application of acephate, estimated to be .75 lb active ingredient/acre. This is astonishing.

Several assumptions do not depend on acephate data, e.g. the assumption that the person exposed weighs 70 kg (children are never mentioned in the entire risk analysis even though they have higher surface to volume ratios, increased permeability of skin, thinner blood-brain barriers, and higher rates of consumption of water), the assumption that exposure is 8 hours for one day, the assumption that meat animals will be exposed in the worst case to only 1/3 of the rate of spray application (it is assumed that 2/3 of the spray will be intercepted by foliage, based on two studies in the forest even though acephate has been and will be used over urban and rural nonforested areas), the assumption that the 70 kg person will drink 2 liters of exposed water for one day, eat .5 kg of exposed meat one day, and eat .5kg of exposed fruits and vegetables one day.

Other numbers used in the scenario do need acephate data and where such data are missing (which, in this scenario, is every instance), estimates have to be made based on data from chemically similar pesticides (no such data are used in this scenario), chemically dissimilar pesticides (a few instances of this occur), or no pesticide data at all (instances of this occur). Examples:

a) Assumption: 10% absorption rate for directly exposed human skin. Basis: No laboratory data for any of the 4 insecticides, so the 10% is derived from two unpublished studies on urine samples following exposure to carbaryl. (F-17)

b) Assumption: 0.3 mg/hr exposure. Basis: No acephate data. One study reported this exposure to a bystander from carbaryl applications to an orchard. (F-18).

The final number derived for human "Direct" exposure, based on 8 hrs, 70 kg, 10% absorption, and 0.3 mg/hr is .004 mg/kg/day. The risk analysis then states, "However, exposures could be as high as those for a project observer (0.029 mg/kg/day worst case) if a resident was outdoors and received a direct application."

F-18. Despite this .029 figure possibility, the .004 figure (i.e. 7X less) stands as the Worst Case - Direct estimate.

c) Assumption (for estimating human dietary consumption of the worst-case animal, rabbit): 10% absorption by the rabbit of spray. Basis: None given.

d) Assumption: 100 ppm residues on vegetation eaten by rabbit. Basis: No acephate data. "Studies of residues on vegetable crops or grass illustrate that initial residues of insecticides range from 1 to 100 ppm. [several references given]." Comment: This assumption is worth noting (see Assumption "i"), because when human dietary exposures via eating exposed fruits and vegetables are estimated, 5 ppm, not 100 ppm are estimated. The rabbits' plants have 100 ppm, the humans' plants have 5 ppm.

e) Assumption: The rabbits eat vegetation with these 100 ppm residues one day. The following rather remarkable statement is made: "Since insecticides are usually rapidly degraded...or, if not degraded, translocated to often inedible plant parts, exposure to animals by ingestion of plants will be only a short-lived phenomenon....[R]esidues degrade to nondetectable levels within 10 to 14 days on vegetation except for grass which can have detectable residues for up to 28 days." F-22.

Comment: Acephate, according to an EPA report (Ghassemi, et al. 1981), penetrates plant tissues quickly. About 9 % of the absorbed acephate is metabolized to methamidophos, a more acutely toxic insecticide. A study by Chevron Chemical Company indicates that Orthene (acephate) is adsorbed onto and/or absorbed into leaf surfaces.(Chevron 1973) Half-lives of 10 days have been reported for acephate and methamidophos in the rinds of various citrus plants sprayed with acephate (Nigg 1979). The assumption that acephate is translocated to inedible plant parts and is a short-lived phenomenon is wrong.

f) Assumption: 10% of the absorbed insecticide reaches tissues. F-28. Basis: None offered.

g) Assumption: 707 ppm residues in water. Basis: Theoretical, based on assumption that an application is direct and there is no incoming water.

h) Assumption: Acephate residues persist in water for a maximum of 4 days. Basis: No acephate data are cited. Studies with carbaryl and trichlorfon are cited. Comment: Acephate does not disappear in 4 days. It has a half-life in water of 16-66 days and methamidophos has a half life of 9-108 days (Tucker and Stephens 1978). A study of persistence of acephate in pond water and creek water held at 9°C recovered 83% of the acephate from pond water after 42 days and 45% from the creek water after 50 days.

i) Assumption: Residues of acephate on fruits and vegetables for human consumption will be 5 ppm. Basis: "...no data were available for insecticide residues on fruits or vegatables [sic] resulting from the use of insecticides to control gypsy moth, residue data from agricultural applications was used as a surrogate." The data used are for carbaryl and trichlorfon showing residues can range from 10-50 ppm but degrade to nondetectable values within 1 or 2 weeks and data for carbaryl that "simple washing" removes 90% of carbaryl residues. "Therefore, vegetables were assumed to have an initial range of residues from 1 to 5 ppm after washing, and these residues degrade to zero (undetectable) within 14 days."

Comment: This lumping together of all four insecticides is unacceptable. I have indicated in Assumption "e" that acephate does not degrade to zero in 1 or 2 weeks. A Chevron study using lettuce, broccoli and cotton leaves found that only an average of 5% of the applied Orthene (acephate) could be washed off leaves 3,7 and 14 days after treatment (Chevron 1973). Therefore, the theoretical 50 ppm residues of acephate on food plants cannot be arbitrarily reduced 90% to 5 ppm. Remember (Assumption "d") that rabbits are presumed to eat vegetation with 100 ppm.

j) Assumption: In the worst case, a double amount of insecticide or two applications would be mistakenly applied to an area. Comment: According to a Pennsylvania report, a mixing error in 1983 resulted in 2X the amount of Dimilin applied/acre in a state park (Pennsyvania Department of Environmental Resources 1983). In other words, the worst case assumption did occur.

3. Discussion of nongenetic effects of the four insecticides in this risk analysis is limited to whether a calculated exposure approaches the ADI or NOEL. Nongenetic effects unique to any of the four insecticides are not mentioned (although they are for the fifth insecticide, B.t. in the main body of the FEIS - insert for p. 64) except for vague references to cholinesterase inhibition for acephate, trichlorfon, and carbaryl. The numbers derived via the exposure scenarios are placed alongside ADIs and NOELs and if they can be juggled so as to stay below either of those numbers, the risks are considered acceptable.

Having developed the entire scenario of resident exposure (the "Direct" exposure category is residents) via the Direct and Dietary doses without any acephate data, the EIS goes on to compare the resultant number (.093 mg/kg/day) with acephate's ADI and NOEL (remember that acephate's NOEL is based on an incomplete data base - see p.4):

The Acceptable Daily Intake (ADI), normally 1/100 of the NOEL (an arbitrary factor of 10 to account for laboratory animal-human variation in sensitivity and a factor of 10 to account for variation in sensitivity among humans), is, in the case of acephate, only 1/10 (F-52). No explanation is given for

this. (The arbitrariness of an ADI itself is illustrated by the fact that EPA issues an ADI for carbaryl of 0.1 mg/kg/day while the World Health Organization considers the carbaryl ADI to be 0.01 mg/kg/day, or one-tenth as much.)

The general public, via four worst case scenarios including Direct and Dietary, exceeds the ADI for acephate by up to 5 times. The Direct and Dietary scenario exceeds it by 4 times. Since acephate's ADI is only 10 times less than the NOEL instead of the EPA's usual practice of placing an ADI 100 times lower (F-52), that gets the general public only two times less than the NOEL for nonhuman animals, i.e., without considering a factor of 10 for intra human sensitivity differences or a factor of 10 if humans are more sensitive than the test animals.

Now, says the EIS (F-70), "The question that must be answered is whether or not these doses exceed an acceptable margin of safety." For acephate, the risk analyst points out that if you take the next higher NOEL (0.75 mg/kg/day as opposed to 0.25 mg/kg/day), then you "approach the 10 safety factor" used by EPA to get acephate's ADI. In other words, the acephate ADI is surpassed by the calculated worst case resident dose, and is only 2 times less than the NOEL, so we take the next higher NOEL (which is, therefore, not a NOEL at all) and see if the calculated dose is 10 times below that even though a factor of 100, not 10 is the one usually used to derive an ADI. This mathematical juggling is unacceptable and reveals the risk analysis for what it is: an apology for chemical insecticides.

Then , says the risk analyst, the NOEL for acephate is for cholinesterase inhibition and "the cholinesterase inhibition NOEL for [acephate] is at least 10 times lower than the NOEL for more pronounced systemic effects and greater than 100 times lower than the NOEL for teratogenic effects. This suggests that the doses in question are still within an acceptable margin of safety when considering major irreversible human health effects [emphasis added]." F-70.

Finally, on F-71 and F-72, the risk analyst concludes the Discussion of Nongenetic Responses - All Exposure Scenarios by saying, "After reviewing the toxicological data base, and the data points and worst case assumptions made to carry out this risk analysis it is clear that: (1) all of the doses in the analysis, including those in question, represent overestimates; (2) even the worst case doses in question are probably still within acceptable margins of safety; and (3) finally, the probability of the general public and occupationally exposed individuals receiving worst case doses is 1.8 X 10 -3 or about 1 worst case dose for every 55 realistic doses received."

Comment: Judgments of acceptable risk are improper in a worst case analysis (or any risk analysis). Acceptability of risk is a political decision, not a scientific or mathematical endeavor. All such references must be removed from the risk analysis.

A critique of the exposure scenarios similar to the above could be carried out with any of the other three pesticides similarly treated. For instance, diflubenzuron is assumed to bioaccumulate 1.0 times in fish, but one study found that it bioaccumulated 13X in the meat, 100-134X in the viscera, and 60-66% of the diflubenzuron had been metabolized to 4-chloroaniline, related to human bladder carcinogens (Booth, et al. 1976). The analysis assumes all plant residues will be zero after 14 days and yet a study has shown that thirty to sixty days after treatment, as much as 90% of the intact pesticide can be detected on leaves (Willcox and Coffey 1978).

III. The Discussions of Accidental Exposures, Sensitive Populations, Synergism, Cumulative Effects, and Carcinogenic Risk of N-nitrosocarbaryl are Toxicologically Incompetent

The analyses of the risks posed by accidental exposures, synergism, and cumulative effects and the risks for sensitive populations are cursory, toxicologically incorrect, and biased.

1. ACCIDENTAL EXPOSURES. In discussing the potential exposures associated with accident scenarios (Tables 11-14 where dermal LD_{50}s are considered for dermal exposure and NOELs and ADIs are considered for water consumption), the analyst merely concludes that the dermal exposure for all four insecticides are near or (in the case of diflubenzuron) many times below the highest level tested (with no deaths) for the dermal LD_{50}s. For acephate, carbaryl, and trichlorfon, near the hgihest LD_{50}s tested, the analyst concludes, "It is not known what effects, if any, could result from these exposures, so it is appropriate to identify these exposures as being hazardous." F-72.

According to EPA's Recognition and Management of Pesticide Poisoning (Morgan 1982), acephate and trichlorfon are moderately toxic organophosphates, which class of pesticides is "efficiently absorbed by inhalation, ingestion, and skin penetration." Poisoning by organophosphates has resulted in peripheral neuropathy that can persist for years. "Organophosphates associated with these chronic illnesses have included some whose acute toxic potential is low [emphasis added]; i.e., there appears to be no relationship between acute toxicity and the likelihood of a chronic neuropathic effect....Other unuusual properties of specific organophosphates may render them more hazardous than basic toxicity data suggest."

A 1979 report by Clement Associates Inc. for the U.S. Department of Labor concluded that children should not be allowed to harvest strawberries and potatoes in fields that had been treated with carbaryl (at rates of 2 and 1 pound per acre, respectively) until 40 days had passed. This would

appear to be a different estimate of the potential for hazard of carbaryl at even non-accidental exposure rates (Clement Associates, Inc. 1979).

The discussion of the hazards posed by being accidentally doused by acephate or trichlorfon (or carbaryl, a carbamate) is clearly inadequate. Any doctor who has treated farmworkers poisoned by exposure to organophosphates and carbamates would have been able to help this risk analyst approach reality about the hazards of being doused by acephate, trichlorfon, or carbaryl. The section must be redone to include at least rudimentary information about hazards of exposure to these pesticides.

2.SENSITIVE POPULATIONS. To account for sensitive individuals, NOELs are reduced by 100, but ADIs are left as they are: "ADIs already include a safety factor (100 in most cases) which was incorporated, in part, to account for differential repsonses within the population." F-79. Comment: In other words, the risk analyst makes NOELs into ADIs and then dismisses the exceeding of ADIs just as was done on F-70.

I do not know the literature on sensitive populations, but at least a cursory review of the literature could have indicated whether carbamates, organophosphates, or substituted ureas were especially suspect. It is a characteristic of this risk analysis to never refer to case reports in the medical literature or any of California's pesticide poisoning reports. I am not familiar with the literature on carbaryl, but I do recall a Union Carbide representative at a gypsy moth conference admitting that some proportion of the general population experiences respiratory or allergic reactions to carbaryl aerial spraying. Even if this were not a correct recall, the worst case analyst owes it to the decisionmaker to have researched the literature surrounding the use of carbaryl in general populations. Otherwise, the decisionmaker will be surprised to learn that a proposed spray program will be opposed by the public because it has been shown that this or that adverse impact has been noted clinically in similar spray operations. This risk analysis exists in some never-never land of numbers, not the reality that first prompted case law to require agencies to consider the risks of their programs.

The Sensitive Populations discussion continues (F-80) on the note that when teratogenicity NOELs are lowered, worst case doses for trichlorfon and carbaryl for the general population are less than the safety factor of 100 used to establish ADIs, but "they are still above the level of 10 which has traditionally been used to account for intraspecies variability [i.e. sensitive individuals]."

COMMENT: This picking which part of the arbitrary 100-fold safety factor that accountrs for intra- and inter-species variability to apply at any one point is unacceptable. For

instance, if carbaryl exposure approaches the NOEL of teratogenicity, leaving only a safety factor of 10, maybe that safety factor of 10 is the one for inter-species variation and the safety factor of 10 for intra-species variation (i.e. sensitive populations) is the one that was "used up." Why can't the risk analyst simply say that worst case exposures of the public to carbaryl may pose risks of birth defects and that is one of the drawbacks that needs to be considered when proposing the use of carbaryl? That is what 40 CFR 1502.22 is requiring.

3. SYNERGISM. The risk of synergism from the presence of these insecticides and others or the simultaneous use of two of these insecticides is dismissed as follows: "For these small comparative doses, a synergistic effect is not realistically expected (Crouch et al. 1983). EPA apparently came to the same conclusion, because they issued a Notice (PR Notice 82-1) on January 12, 1982 (US EPA 1982b), rescinding the requirement for submission of tank mix compatability data. The Notice stated that EPA had examined considerable data and found no evidence of potentiation involving pesticides." x

While this risk analysis cannot be expected to come up with numbers for precise estimates of synergistic effects of these four pesticides and the other pesticides present in the environment (in part because there are giant gaps in such information), it can at least indicate that an understanding of synergism is grasped. Tank mix compatibility is not what people have in mind when they speak of health risks of synergism. Three references immediately come to mind that have documented the synergism of pesticide toxic effects by carbaryl (Statham and Lech 1975a, 1975b, 1976). The California Department of Food and Agriculture requires a longer safety interval when two or more organophosphates have been applied because organophosphates are particularly prone to synergistic effects. Crocker (1976) experimentally demonstrated the interaction of ubiquitous insecticide carriers with virus to increase viral lethality and effect on the liver and central nervous sytem of mice. The discussion of synergism must be redone.

4. CUMULATIVE EFFECTS. The discussion of cumulative effects is covered in three paragraphs (F-82) and is limited to a discussion of whether someone would ingest too much of any of the insecticides, given that other food may have residues of them. The answer given is that a person might accumulate a dose that would exceed the ADI by eating food cotaining the maximum federal tolerance in addition to gypsy moth spray program exposure, but the lowest NOEL or teratogenicity NOEL would not be exceeded. y

Comment: The risk analysis clearly fails to deal with cumulative effects of pesticides in general. Whose duty should it have been to recognize the risk in using massive amounts of DDT for agriculture in regions that were also using DDT to

control malaria-carrying mosquitos? Resistance to DDT and other pesticides among the mosquitos is now leading to a resurgence in malaria. Whose duty is it to be concerned about the accumulation of pesticides in drinking water? If each risk analysis concerns itself with the risk of contracting cancer from the cigarette smoke in one executive room, whose duty is it to recognize that smoking is going on elsewhere, too? Again, the analyst cannot be expected to determine the quantitative relation of each of these pesticides to general pesticide use in the society, but a brief examination of which, if any of the pesticides considered, is likely to pose cumulative problems is essential.

We have been told, for instance, that the second of two diflubenzuron applications for Oregon's gypsy moth program will have cumulative effects on the gypsy moth. The first application will not have disappeared from the foliage 14 days later, and the second application will increase the effect against the gypsy moth. If this effect is a plus on the side of efficacy for diflubenzuron, it would perhaps be a minus on the side of cumulative effects for humans. This is the kind of trade-off decisionmakers need to understand. z

5. CARCINOGENIC RISK OF N-NITROSOCARBARYL. The section on carbaryl's carcinogenic risk is not adequate. If, as it appears (F-11), the lifetimes of humans and rats are being equated, this is in error. There is much evidence that carcinogenic response is proportion to the dose per unit body weight and not to lifespan. In other words, if 10 gm/kg applied during 6 months gives rise to tumors in rats in 1 year, then that same dose to another susceptible species will give rise to tumors in about 1 year. As the dose rate or total dose decreases, the length of time to development of tumors increases. Exposure to low doses puts a long lived animal (e.g. humans) at risk for tumors at the end of life. aa

The single study used for calculating the "potency" of n-nitrosocarbaryl as a carcinogen is that by Eisenbrand et al. (F-11). In this experiment the animals were fed such high doses that most of them may have died before only 29% got cancer. Another study must be considered which will change the estimate of risk from cancer: that of Lijinsky and Taylor (1976) in which 25X less of a dose resulted in 75% of the animals contracting cancer. The results following administration of a large dose over a short period (as in Eisenbrand's study) give results that are grossly in error in comparison to Lijinsky's study.

In addition, children are at particular risk for cancer from N-nitrosocarbaryl both by virtue of greater sensitivity to carcinogens and a longer life span following exposure.

Finally, since the kinetics of formation of nitrosocarbaryl in the inhomogeneous milieu of the stomach are unknown, the yield of nitrosocarbaryl might be orders of magnitude higher than predicted from a reaction of dilute solutions in a flask. bb

This section must be redone in light of the study by Lijinsky and Taylor and in light of children. It is indefensible otherwise. A simple reading of Lijinsky's affidavit in OEC v. Kunzman (seeing as how this worst case analysis purports to address issues raised in OEC v. Kunzman and not NEPA) would have resulted in a more toxicologically acceptable discussion of the risk posed by spraying carbaryl.

I have not looked at the trichlorfon discussion carefully. Perhaps it is similarly in error.

6. MISCELLANEOUS. In the discussion of Risk of Heritable Mutations (F-77), the following is stated: "Trichlorfon is a more potent mutagen than the other three insecticides under consideration, testing positive in all tests except the whole animal, mouse micronucleus test, reviewed by Jones et al. (1984). There is also suggestive evidence that it can reach germinal tissue. However, even if trichlorfon causes heritable mutations in humans, it's unlikely that any heritable response would be measureable."

cc

This is primarily because of the genetic variability, the small number of offspring and the long generation times in humans, and because of the relatively low short-term exposure that would occur from trichlorfon use against gypsy moths....If a mutation occurred, it would be impossible to determine whether it was caused from pesticide exposure or exposure to one or many of the natural mutagens known to exist in our environment."

Comment: The point of a risk analysis is to determine whether an adverse helath or environmental effect might occur, not whether it will be measureable. We will never be able to trace a specific person's lung cancer to the fact that she smoked three packs of cigarettes a day, but numerous people nevertheless refrain from smoking on the grounds that it increases their chances of contracting lung cancer. By the same analogy, if all these genotoxicity tests are waving warning signs that trichlorfon is genotoxic, then to say "Oh, well, you won't be able to measure it" is inexcusable. Is this risk analysis the attempt of an insurance company to see whether it can be sued for aspray program's results, or is it an exercise in trying to warn of risks involved with various gypsy moth control tools?

In the summary at the front of the EIS (p. 23), it is mentioned that "mitigation measures should be taken to minimize exposure to trichlorfon, especially to genetically sensitive individuals. [Are you genetically sensitive? Are your children?] One method for limiting exposure would be to use trichlorfon only in sparsely populated areas. This would not reduce exposure to individuals in the treated area, but it would reduce the probability of heritable mutations occurring."

Comment: This recommendation should go over big with rural people. Another method for limiting exposure would be to not use trichlorfon.

IV. CONCLUSION

This worst case analysis needs to be redone so that a decisionmaker can compare all five insecticides (including B.t.) on potential human health risks. Perhaps the most helpful would be to compose a chart for each insecticide with the headings "Consequence", "Credible Source(s) of Concern", "Probability of Occurrence", and "Source(s) of Probability Calculation". dd

This chart would have several advantages over the present risk analysis:

(1) It would carefully separate the consideration of consequences from the probability of their occurrence.

(2) It would warn the decisionmaker of the major concerns that will be raised by biologists, ecologists, health professionals, and citizens when and if the decision to spray pesticides is made.

(3) It would warn decisionmakers of the "softness" of some of the proability calculations. As seen above, for instance, the calculation of risk of nongenetic effects of acephate exposure is produced without one piece of experimental or field data on acephate.

(4) It would reward the production of useful and needed information, because it would lay out in a clear fashion where data gaps exist. For instance, given that apparently nobody knows the rate of dermal absorption for any of the four insecticides, the chart would have to indicate that in its column on "Source(s) for Probability Calculations" of general public and worker exposure hazards. The various data gaps would be laid out and cost-effective decisions could be made as to which data gaps would not be exorbitantly expensive and/or which are most crucial for future research. This would aid in the fulfillment of the terms of 40 CFR 1502.22 whereby data gaps are supposed to be filled before a worst case analysis is drawn up unless filling those gaps is exorbitantly expensive. Filling all the data gaps will always be "exorbitantly expensive." At some point, someone has to take responsibility for filling some of them.

(5) It would allow the public (professional or lay) to more easily plug in documented information that the risk analyst had inadvertently (or otherwise) missed. It would show, for instance, that the calculation that acephate residues would disappear in four days from water was based on no data and that the analyst

had missed studies that indicate that acephate (and its metabolite, methamidophos) can be extremely persistent in water.

(6) It would allow for admitting that the probability of certain hazards cannot be calculated with any degree of certainty, but that the risks nevertheless exist and the decisionmaker will have to take that into account when making pesticide decisions. In other words, if the column on "Probability of Occurrence" of genetic damage by trichlorfon has to remain blank, then the decisionmaker will have to take that into consideration.

A debate is currently going on in public (and private) arenas as to the reasonableness of 40 CFR 1502.22. The worst case analysis promulgated in the Draft Supplement is inadequate, not because it is beyond the abilities of the federal agency to produce an adequate one, but because the USDA is unwilling to admit that pesticides are hazardous and so makes complicated a rather straightforward requirement: Consider the risks involved in using pesticides. The public's concern over widespread use of chemical insecticides as the government's tool of first choice for pest control on public and private lands will not go away by making the risk analyses ever more convoluted and tortuous. Face the fact that pesticides carry risks and then make pest control decisions in light of the fact that documentation of the risks is available, the probabilities of occurrence are often impossible to determine, and a pesticide that may be just what one would want to use to kill a single pest may therefore, in the real social and political world, have to remain unused because of the adverse impacts it may have on the environment and/or public health. If the decision is made to employ the pesticide, that decision will carry with it explicit recognition that risks are being taken. We all make such decisions in our everyday life.

Sincerely,

Mary H. O'Brien

Mary H. O'Brien
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and for Citizens for Safe Control
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February 1, 1984

COMMENTS OF THE PESTICIDE HAZARDS CLEARINGHOUSE ON THE U.S. DEPARTMENT OF AGRICULTURE GYPSY MOTH SUPRESSION AND ERADICATION PROJECTS DRAFT SUPPLEMENT TO THE FINAL ENVIRONMENTAL IMPACT STATEMENT (1985)

Kenneth Hobbs, Research And
Publications Director

These comments on the Draft Supplement To The Final Environmental Impact Statement (abbreviated to DS herewith) are preliminary. The Pesticide Hazards Clearing House (PHCH) plans to write more extensive comments with full documentation of references when the Final Supplement To The Final Environmental Impact Statement (abbreviated to FS herewith) is published.

Comments are also planned on the Final Environmental Impact Statement (1984) in the future.

The PHCH is a non-profit organization which acts as a clearinghouse for information on hazards of pesticides. Information from all sources is welcomed and will be added to the files.

PART A -- FACTUAL ERRORS IN THE DRAFT SUPPLEMENT TO THE FINAL ENVIRONMENTAL IMPACT STATEMENT

17, Paragraph 1, Line 14 -- "cholinesterase" is mispelled. a

F-7, Paragraph 2, Lines 3-4 -- "mouse forward mutation assay" should be reworded to "mouse lymphoma cell culture forward mutation assay". b

F-8, Paragraph 1, Line 2 -- The test microbes are mispelled. The corrections are as follows: "Salmonella typhimurium", Escherichia coli" and "Saacharomyces cerevisiae". c

F-8, Paragraph 2, Line 2 -- "Simmon" is mispelled. d

F-8, Paragraph 2, Lines 10-11 -- The Degraeve et al. ('81) reference is an abstract which erred in reporting the results of these studies. The full studies were published after this abstract was published, & they found negative results for cytogenetic effects of trichlorfon in mouse bone marrow cells & spermatogonia, & also in the dominant lethal assay(23). e

F-9, Paragraph 3, Lines 5-6 -- The Doull ('62) study used trichlorfon of approximately 95.3 % purity (the purity of Bayer/Mobay Chemical Company trichlorfon in the late '50's and early '60's)(24). Since there are a number of mutagens among the manufacturing impurities of trichlorfon(25), it's necessary to use trichlorfon of equal or greater purity to the chemical now being produced (99.1% purity)in studies of its mutagenic/teratogenic/carcinogenic effects (26). f

F-13, Paragraph 1 -- Obviously, you can't make the assumption that the rate of malignant tumors in the control group is zero when , in actuality, it is 7.5%. Thus this whole analysis is invalid. g

F-15, Paragraph 3, Lines 8-10 -- The DS states that urinary 1-naphthol was measured in the studies cited in this paragraph. Actually, 1-naphthol is not found intthe urine after carbaryl absorption. These metabolites are converted back to 1-naphthol in the analytical technique, which is then measured. h

F-15, Paragraph 4 to F-16, Paragraph 1 -- The data from the Schulze et al. ('79) study [the DS erroneously cites this as "Schulze (1979)"] on urinary 1-naphthol for spray pilots, mixers and loaders is unusable because "virtually all" of the pilots flew other carbaryl spray jobs on farms after the early morning gypsy moth spraying (Schulze et al., 9). Since the data for pilots is not separated out from the data for mixers and loaders none of the data can be used to asses exposures from gypsy moth spray projects which are almost always done in the early morning. i

F-15 to F-19 -- The data for urinary 1-naphthol in project workers from the SCESC studies must be corrected because they typically did both early morning and evening applications, and aerial gypsy moth spray projects are almost always done in the early morning. j

The applications were made from 5AM-8:30AM and from 7PM-9PM, with the total application hours during a day being 5 1/2. Thus, the percentage of spray exposure time accounted for by the morning projects was 63.6% of the total daily exposure time. This percentage is used to set a correction factor of .636, by which one multiplies the urinary 1-naphthol figures in order to get corrected realistic exposure doses. We will do this in our comments on the FS.

F-18, Paragraph 2, Lines 11-16 -- In the calculation of the estimated realistic exposure dose for a resident with 247 ppb urinary 1-naphthol the multiplier wasn't used. Using the multiplier increases the realistic exposure from .0017 to .0050 mg/kg. Since this value was later converted to the estimated value for a person 250 feet from the treatment area, you also have to correct the latter value to .0034 mg/kg (F-19, Paragraph 2). k

F-25, Paragraph 3, Line 9 -- "metabolites" is mispelled l

F-26, Paragraph 1, Line 8 -- "soluble " is mispelled m

F-60, Lines 3-5 -- The in vitro yield of N-nitrosocarbaryl from the test tube reaction of carbaryl at 100 uM and sodium nitrite at 500 uM (the lowest concentrations used) is 1% of the maximum yield theoretical yield. This data is from Eisenbrand et al.,(1975). n

Substituting the correct value in the DS analysis is necessary.

F-84, 5th Reference -- "nitrosomorpholine" is mispelled o

F-84, 8th Reference -- "ascorbate" is mispelled p

F-84, 11th Reference -- "mutagenicity is mispelled q

F-86, 5th Reference -- "teratogenic" is mispelled r

F-86, 10th Reference -- "guthion" is mispelled s

F-87 , 4th Reference -- substitute "Schulze, T.L., et al." t

F-89, 13th Reference -- "Symposium" is mispelled. Also insert "American Chemical Society, Washington, D.C" after the given reference. u

F-89, 14th Reference -- eliminate "ca.", and insert " Volume 3". v

F-91, 1st Reference -- Under "NOEL" note that the two doses given are for two separate studies. Under "Reference" note that "Teratogenic " is mispelled. w

F-91, 2nd Reference -- Under "Type Of Test" insert "maternal weight loss observed at 200 mg/kg/day, but not at 150 mg/kg/day". Under "NOEL" add that non-statistically significant effects (omphalocele) were observed at 150 mg/kg/day (see DS, 17, paragraph 1). Under "Reference" note that "teratogenic" and "gavage" are mispelled. **x**

F-91, Reference 5 -- Under "Type Of Test" insert "Mutagenic/ Teratogenic (3 Generation Reproduction Study) , Combined With A Male Dominant Lethal Assay". **y**

F-91, 3rd Reference -- Under "Type Of Test" add "Mutagenic/ Teratogenic (3 Generation Reproduction Studies)". Under "NOEL" add "200 mg/kg/day in one 3 generation reproduction study". Also add "10 mg/kg/day (LOAEL) for the other 3 generation study ". [On p. 394 the authors note that there was a 42% increase in incidence of dilated uteri in the third generation first litter pups]. **z**

F-91, 4th Reference -- Under "Type Of Test" add "Teratogenic". **aa**

F-91, 6th Reference -- Under "Type Of Test" add "Teratogenic" and "(missing internal organs)". **bb**

F-91, 7th Reference -- Under "Type Of Test" add "birth defects, neonatal functional deficits [decreased survival and weight gain up to weaning] and behavioral anomalies [excessive crying]". Under "NOEL" add "6.25 mg/kg/day LOAEL for birth defects; 3.25 mg/kg/day LOAEL for neonatal functional deficits and behavioral anomalies" **cc**

F-92, 1st Reference -- Under "NOEL" delete"50mg/kg/day" and insert "1000 mg/kg/day". **dd**

F-94, Last Reference -- Omit this reference since it used Russian manufactured trichlorfon of uncertain purity (cf. Comments on the DS, paragraph 6). **ee**

ff

F-95, 4th Reference -- Under "Type Of Test", delete "(Low Fetal Weight)". Under "NOEL", delete "400" and add "300" for the "Hamster" data line.Also,for the "Rat" data line delete "432 (LOAEL)" and substitute "480 (LOAEL)". For the "Mouse" data line substitute "400; 500 (LOAEL)".

Under"Reference" note that "gavage"is mispelled.

gg

F-95, 4th Reference -- Add the following data line: "Rat; Teratogenic; 375(NOEL)/432 (LOAEL); R.E. Staples, R.G. Kellam , J.K. Haseman, "Developmental Toxicity In The Rat After Ingestion Or Gavage Of Organophosphate Pesticides (Dipterex, Imidan) During Pregnancy", Environmental Health Perspectives, 13: 133-140, 1976".

F-95, Footnote -- Delete "Pesticides" and "(ca.)".

PART B - OTHER COMMENTS ON THE DRAFT SUPPLEMENT TO THE FINAL ENVIRONMENTAL IMPACT STATEMENT

F-3 to F-5, "Review Of Toxicological Studies; Background Information" -- On p. F-4, paragraph 1, lines 8-12, the DS notes the types of studies chosen for inclusion in the "Review". Intraperitoneal injection studies (studies injecting the test chemical into the intraperitoneal cavity) are also appropriate because the test substance immediately enters the portal circulation. In this sense, these studies are similar to oral studies where the substance enters the portal circulation(which empties into the liver)after absorption from the glandular stomach and the small intestine mucosa(16). On the other hand, the metabolic effects of the enzymes in the mucosa on ingested chemicals aren't operative after intraperitoneal injection.

hh

Also, in the case of mutagenic chemicals that are detoxified in the liver such as carbaryl, acephate and trichlorfon, oral and intraperitoneal injection may result in an underestimation of the actual mutagenic potency from low dermal or inhalation exposures. The reason for this underestimation is that dermal or inhalation exposures result in the chemical directly entering the bloodstream, and the chemical potentially may travel to any part of the body without first entering the liver where it is apt to be detoxified (17).

Thus, with weak, easily detoxified mutagens such as trichlorfon when the only data available is oral or intraperitoneal studies one often has to rely on <u>in vitro</u> (microbe or cell culture) mutagenicity studies in conjunction with data on their detoxification properties in bodily fluids to assess their hazards.

F-5 to F-6, "Nongenetic Responses" -- The DS errs in its assumption that all teratogenic effects (birth defects due to chemical exposure during pregnancy) are "nongenetic responses", and consequently have "no effect doses" at some low level of intake.

ii

Recent research has shown that morphological birth defects such as major skeletal defects (eg. missing bones, extra bones) may be due to chromosome breaks or exchanges(18). Certain aberrant hair color spots are due to gene mutations (19). Research is also being done on the probable role of chemical mutagens in inducing fetal enzyme anomalies(20) or behavioral anomalies through gene mutations or other types of mutation(21).

jj

F-6 to F-8, "Genetic Responses: Mutagenicity" -- RE mutagenicity, one should bear in mind that a spectrum of different types of mutations normally occurs at different doses(*).

At higher doses chromosome breaks or exchanges between broken chromosomes may be induced by mutagenical chemicals. These mutations are referred to in the DS as "chromosome aberrations", "structural chromosome aberrations", "chromosomal effects", "chromosomal damage" or "chromatid-type aberrations". Some animal tests for these mutations are the micronucleus test which detects chromosome breaks, and the dominant lethal assay which detects sperm chromosome breaks causing death to the animal's offspring before birth(14).

At lower doses a chemical may not induce chromosome breaks or exchanges, but may induce gene mutations in which

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* -- The novice who's confused by this discussion & has taken high school biology and chemistry should purchase an introductory text on genetic toxicology such as D. Brusick, Principles Of Genetic Toxicology, '80. Reference to a medical dictionary at one's local public library is also recommended.

nucleotide base pairs (the building blocks of DNA) are altered so that (1)their normal order in the DNA molecule is altered, or (2)they are added to an area of the DNA molecule where they wern't found before, or (3) they are deleted from the DNA molecule. The DS refers to gene mutations as "mutations", "forward mutations" or "reverse mutations".

At very low doses such as those commonly found in the general environment a mutagenic chemical may not induce mutations at all, but can exert a possible co-mutagenic effect due to its contributing to the saturation of mutation repair enzymes (DNA repair enzymes). The depletion of these enzymes may increase the chance that other mutagenic chemicals found in the body simultaneously with the mutagenic chemical we are looking at may induce chromosome breaks, chromosome exchanges or gene mutations(7). Tests for DNA repair (unscheduled DNA synthesis), primary DNA damage or sister chromatid exchanges indicate the capacity of a chemical to bind to DNA thus triggering repair enzymes that remove it from the DNA & repair any damage left afterwards.

In order to clarify these spectra of mutagenic effects for the chemical gypsy moth insecticides the PHCH intends to chart the dose-response curves for each effect measured by particular <u>in vitro</u> test systems. All of the insecticides curves will be on the same chart for a particular test system.

RE the discussion of diflubenzuron mutagenicity, studies are missing on the positive activity of the liver metabolite 4-chloroaniline in: (1)<u>Salmonella</u> (bacteria) and mouse cultured lymphoma cells gene mutation tests; (2)chromosome breakage in Chinese hamster ovary cells in culture and (3)sister chromatid exchanges in Chinese hamster ovary cells. kk

Re carbaryl's mutagenicity, its major mutagenic hazard probably derives from the unmetabolized parent molecule. Unmetabolized carbaryl is the most potent inducer of gene mutations and of DNA repair (unscheduled DNA synthesis) among the gypsy moth insecticides with a similar mode of mutation induction (carbaryl, trichlorfon and acephate). Also, it is the least susceptible to metabolic breakdown and detoxification in the blood among these three insecticides. This means that it has a greater capability to reach:(1) tissues which are most susceptible to mutation, (2)the fetus where it may either induce birth defects or mutations to the fetal pre-sperm or pre-ova cells, or (3)the male germ cells where it may cause sperm mutation(*).

The best evidence to date of carbaryl's possible potential to induce sperm gene mutations is a study which found that it induced an increase of 8% and 10% in malformed sperm after doses of 4 mg/kg (intraperitoneal) or 1.8 mg/kg per day for 7 days, respectively(10).

In our judgment, the epidemiological studies on carbaryl mutagenicity, propensity to induce birth defects or propensity to induce sperm shape or count anomalies are all useless for assessing its hazard to the general population or to spray project workers.

RE trichlorfon's mutagenicity, the major factor to consider is that it is metabolized to a non-mutagenic form in blood much more rapidly than carbaryl, which reduces its relative hazard greatly. Although it has caused a 34% increase in dead fetuses in the dominant lethal assay at 405 mg/kg (intraperitoneal)(12), it is doubtful whether it can reach the male germ cells after much lower doses resulting from spray exposures. The same line of reasoning applies to acephate which has a similar chemical structure and is detoxified to a non-mutagenic form rapidly in the blood.

The Kiraly et al. study of trichlorfon manufacturing workers is useless for assessing the risk of the chemical to other population groups. Also, the DS omits the study finding that

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* -- The DS omits mention of the Bukin & Filatov study(9) which found un-metabolized carbaryl in rabbit testes up to 4 hours after administration of 400 mg/kg (400 milligrams per kilogram body weight) carbaryl by stomach tube (detection limit was 1 part per billion). The study also found carbaryl in the brain, liver, bile & kidney fat 4 days after the administration. The brain is an organ which is susceptible to mutation by chemicals with a mutagenic mechanism similar to carbaryl's(27).

trichlorfon is a weak inducer of chromosome breaks in cultured hamster cells.

F-9 to F-14, "Genetic Responses: Cancer" -- One should bear in mind the mutagenic spectra of these insecticides when assessing carcinogenic risks, particularly in light of recent research showing that chromosome breaks and/or exchanges are necessary to induce benign tumors, while gene mutations can change benign tumors into malignant ones(13).

Knowing this, carbaryl would be expected to have the most potent carcinogenicity among these insecticides. However the data base is inadequate to assess its mutagenicity. The two 2 year rodent studies using Union Carbide Co. product done for Union Carbide are flawed by: (1)inadequate numbers of animals used (20 per dose and sex group); (2)high mortality due to infections affecting both test and control animals and causing many animals to die before they were old enough to develop neoplasms, and making calculations of increases in naturally occuring tumors impossible in the cases where the mortality in the control groups was higher than that in the test groups; and (3)examination of an inadequate number of organs microscopically. Incredibly enough, the EPA has not required that one or both of these studies be repeated with the improved testing methods recently drawn up (DS, F-88, 7th reference)(re the Union Carbide rodent studies see DS, F-88, 5th reference and F-89, 5th reference).

The PHCH intends to get these carbaryl carcinogenicity and review them in greater detail for inadequacies.

RE carcinogenicity studies with high purity trichlorfon manufactured by Bayer/Mobay Chemical Co. (99.1% pure) or its equivalent, Teichmann and Hauschild ('78) found one skin papilloma at the dermal application site out of 30 female mice with none in the controls. Teichmann and Schmidt ('78) found one injection site fibrosarcoma out of 25 intraperitoneally injected female golden hamsters, with none in the controls. The PHCH intends to review the Teichmann group studies more closely, along with a recently finished 90 week mouse study done by Bayer/Mobay. The Teichmann group studies (which also include a 118 week rat study) are flawed by their not routinely examining all the organs microscopically.

F-14 to F-19, "Analysis Of Exposure: Human Exposures And Estimated Dose Levels" -- We think there are some serious methodological flaws in the exposure analysis for carbaryl and the other insecticides for which the carbaryl exposure analysis is used. These result in an underestimation of the estimated human exposures to the sprays.

The analysis uses separate methods to estimate exposures to carbaryl after either "realistic exposure " or "worst case exposure".

oo For the estimation of realistic exposures total absorption of carbaryl is calculated from measurements of the metabolite 1-naphthol in the urine of people exposed to carbaryl in spray projects, multiplied by a multiplier. However, the analysis appears to underestimate absorption due to the use of an incorrect multiplier. The multiplier, 3, is derived from a study cited on F-16, paragraph 1 which found that 32.8% of a carbaryl dose in human studies (?) is excreted as 1-naphthol or 1-naptholmetabolites by 12 hours after dosing.

We have searched the literature and the only human dosing study we are aware of, Knaak et al.('68),(2) determined that 21% of a carbaryl dose was excreted as 1-naphthol during the the first 24 hours after dosing. During the first 12 hours after dosing less that 21% of the dose was excreted as 1-naphthol, since the peak urinary excretion was found 4-12 hours after exposure(3).

Substituting this study for the study cited in the DS results in an increase in the multiplier to less than 5, which increases the estimated realistic exposure value by greater than 67%. We intend to get the study cited in the DS, examine it for validity, and if needs be recalculate the realistic exposure value using the Knaak, '68 data. This will be in our Comments On The FS.

pp The calculation of worst case exposures in the DS involves the theoretical maximum dermal dose to a fully clothed 7o kg man with 2 square feet of exposed skin (he's 80% exposed)(8) by an assumed dermal absorption rate of 10%.

This assumed dermal absorption rate is probably low. Mittleman estimated that factory workers exposed to carbaryl powder absorbed about 12% of the contacted dermal dose(4). Since liquid formulations of carbaryl are more toxic in animals after dermal application than carbaryl powder(5), it seems reasonable to assume that there's substantially greater absorption of liquid formulations such as the Sevin-4-Oil used in most gypsy moth aerial spray projects than there is of carbaryl powder. Thus, the absorption rate should be set higher

than 12%.

The PHCH intends to get the Weil et al. study on the differential dermal toxicity of powder versus liquid formulations of carbaryl (Reference 5 above), and to use it to attempt to calculate a multiplier to convert Mittleman's dermal absorption rate to an estimated dermal absorption rate for liquid formulations. This will make possible the calculation of the estimated dermal absorption from spray projects.

Lastly, in order to verify the use of the 10% assumed dermal absorption rate, the DS makes a rough estimate of the dermal absorption rate by dividing the carbaryl oral rat LD_{50} by the dermal LD_{50}. This method results in an estimated rate of 5.2%.

It seems sensible to criticize this figure as being excessively low, particularly when one considers the Feldman and Maibach ('74) study and other dermal absorption and toxicity studies (see above). This method's apparant underestimation of the actual dermal absorption is probably explained by slower carbaryl absorption into the bloodstream after dermal absorption than after stomach tube ingestion. Consequently, if a certain amount entered the bloodstream by the dermal route - as opposed to entrance by the oral route- dermal administration would probably result in a lower blood concentration over a longer period, while the oral administration would probably result in a higher concentration over a shorter period of time. While the same amount of carbaryl would reach an organ susceptible to carbaryl's lethal effects such as the lungs,the dermal application might be tolerated because the concentration of carbaryl never reaches the level required to induce organ malfunction and subsequent death.

RE F-19, paragraph 2, the analysis considers exposures to spray drift only up to 250 feet from the treatment. However the analysis fails to consider exposures to longer range drift. EPA studies show that 10-40% of aerially sprayed pesticides drift more than 1000 feet from their target, and studies done by Me. Department Of Human Services find that aerially applied Sevin-4-Oil drifts at least 80-100 miles off target(28). qq

Oregonians for Food and Shelter

1270 Chemeketa Street Northeast
Salem, Oregon 97301
370-8092

14

February 1, 1985

Mr. Gary E. Moorehead, Staff Officer
USDA-APHIS-PPQ
Federal Building, Room 663
Hyattsville, MD 20782

RE: Draft Supplement to the Final Environmental Impact Statement for Gypsy Moth Suppression and Eradication Projects.

Introduction

We welcome the opportunity to comment on your draft supplement. Oregonians for Food & Shelter, Inc. (OFS) is a non-profit, tax exempt Oregon corporation. OFS membership includes agricultural, timber, nursery, tree fruit, small business and other pesticide use interests. OFS operates to provide factual pesticide and pesticide use information to its members, supporters and the public.

We have thus read your draft supplement with great interest. An infestation of the gypsy moth in Lane County, Oregon has been identified and the infestation is staggering in both size and density. A quarantine is in effect on the movement of timber, timber products and Christmas trees. A proposed eradication program is designed to end the environmental, residential, liveability and economic destruction that the infestation threatens. Our members are in full accord with the need to eradicate this pest, and our comments are designed to support this position.

Need For The Supplement

A series of Ninth Circuit Court of Appeals and Oregon Federal District Court decisions have called into question past USDA/APHIS NEPA compliance practices. These decisions, in effect, mandate construction of a worst case analysis (WCA) anytime any data gap or scientific uncertainty surrounds a proposed action and poses any possible effect.

We regard this judicial mandate as an unfair reading of the NEPA regulations. The courts are telling agencies with expertise how to manage their affairs on behalf of the public and the management practice imposed is a crystal ball gazing technique. This is an unnecessary waste of agency resources and taxpayer dollars, yet it is the reality we face today.

This supplement, then, is designed to address gypsy moth eradication and suppression program events and effects that are both foreseeable and highly speculative, as required by the courts.

Adequacy Of The Supplement

We believe the supplement more than fulfills the need for the agency program review as required by NEPA.

NEPA commands that federal agencies entertain an environmental and human health review process for certain proposed programs. In simplest terms, that process must include proposed program impacts assessment, program alternatives and no action options.

This supplement clearly identifies for the decision-maker all possible program impacts for gypsy moth eradication and/or suppression projects that may employ a variety of eradication and/or suppression tools. No reasonable decision-maker, or public interest, should ask for more than is provided in the supplement. The draft supplement does fulfill the agency's remaining NEPA duties, as identified, following publication of the final environmental impact statement.

Decision-makers now have before them all realistically expected impacts and a wide range of possible -- but highly improbable -- impacts. That is what NEPA, according to the courts, demands. Decision-makers can make gypsy moth program choices with open eyes. That is all that NEPA requires.

Some parties may continue to quibble that not everything is known about gypsy moth program impacts. That kind of theoretical games playing can be engaged in for any and every activity of the human species. We simply don't know everything about anything -- and that is as it should be as gaining answers to one question necessarily and rightly leads to the asking of new questions.

This supplement provides real world information for construction of safe, efficacious, necessary gypsy moth eradication and/or suppression programs. Nothing more can or should be asked of such program managers.

The supplement makes the USDA/APHIS programmatic environmental impact statement (PEIS) process whole.

Conclusion

The draft supplement for gypsy moth eradication and suppression projects is adequate.

In fact, in our opinion, it can be said that USDA/APHIS has more than fulfilled the program impacts review process mandated by NEPA.

It should be well understood that this is a flexible, dynamic process. As new information arises, reassessments will be undertaken. And, it must be remembered, site specific assessments will add to this process as particular programs are proposed.

What this supplement does, and does well, is complete the USDA/APHIS obligation under NEPA for construction of a PEIS for gypsy moth eradication and suppression programs.

We thank you for your attention and commend you for a job well done.

Sincerely,

David H. Dietz, Program Director
Oregonians for Food & Shelter

DHD/ksp

RCVD. FOSS 2-4-85

FM208 01/25/85

NORTH CAROLINA STATE CLEARINGHOUSE
DEPARTMENT OF ADMINISTRATION
116 WEST JONES STREET
RALEIGH NORTH CAROLINA 27611

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INTERGOVERNMENTAL REVIEW COMMENTS

MAILED TO

US DEPT. OF AGRICULTURE, FOREST SERV.
GARY E MOOREHEAD
FEDERAL BUILDING, ROOM 663 - APHIS - PPQ
HYATTSVILLE, MARYLAND 20782

FROM

MRS. CHRYS BAGGETT
DIRECTOR
N C STATE CLEARINGHOUSE

PROJECT DESCRIPTION

THIS EIS COVER FOREST SERVICE AND ANIMAL AND PLANT HEALTH INSECTION SERVICE GYPSY MOTH SUPPRESSION AND ERADICATION PROJECTS IN THE U.S.

SAI NO 85E00000450 PROGRAM TITLE - DRAFT SUPPLEMENT TO THE FINAL EIS

THE ABOVE PROJECT HAS BEEN SUBMITTED TO THE NORTH CAROLINA

INTERGOVERNMENTAL REVIEW PROCESS. AS A RESULT OF THE REVIEW THE FOLLOWING

IS SUBMITTED (X) NO COMMENTS WERE RECEIVED

( ) COMMENTS ATTACHED

SHOULD YOU HAVE ANY QUESTIONS, PLEASE CALL THIS OFFICE (919) 733-4131.

State of South Carolina

Office of the Governor

RICHARD W. RILEY
GOVERNOR

OFFICE OF EXECUTIVE
POLICY AND PROGRAMS

February 1, 1985

Mr. Gary E. Moorehead, Staff Officer
USDA - APHIS - PPQ
Federal Building, Room 663
Hyattsville, Maryland 20782

Dear Mr. Moorehead:

The South Carolina Project Notification and Review System has conducted an intergovernmental review on the Draft Supplement to the Final Environmental Impact Statement, Gypsy Moth Suppression and Eradication Projects, Forest Service, Animal and Plant Health Inspection Service, United States Department of Agriculture, as authorized by Presidential Executive Order 12372, "Intergovernmental Review of Federal Programs". As of this date there have been no comments received as a result of the review.

The State Application Identifier number for this project is EIS-8501-001. This number should be used in any future correspondence with this office regarding this proposal. The State of South Carolina is appreciative of the opportunity to review this proposed activity. If I may answer any questions, or be of further service in any way, please do not hesitate to contact me.

Sincerely,

Danny L. Cromer
State Single Point of Contact
Intergovernmental Review

National Coalition Against the Misuse of Pesticides
530 7th Street, SE • Washington, DC 20003 • 202/543-5450

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February 1, 1985

RCVD. FOSS 2-5-85

Gary E. Moorehead
Staff Officer
UDSA-APHIS-PPQ
Federal Building, Room 663
Hyattsville, MD 20782

Dear Mr. Moorehead:

This letter shall serve as the National Coalition Against the Misuse of Pesticides' comments on the "Draft Supplement to the Final Environmental Impact Statement" on the USDA Moth Suppression and Eradication Projects. Given the public concern about pesticide spray programs and the attendant risks and unknowns, we find the above cited document to be especially inadequate.

Our concerns can be summarized as follows:

1. The document is extremely difficult to understand and does not enable the public and/or decision makers to comment intelligently. Our understanding is that a document of this import should, according to Council on Environmental Quality regulations, be readable and intelligible. a

2. The summary does not fully reflect the general findings of the Environmental Impact Statement. This, too, precludes adequate public participation. b

3. The document fails to discuss fully and equally the potential health effects related to the range of proposed suppression and eradication programs. c

4. In limiting comments at this time to the Draft Supplement, the government has blocked the public's right to comment on the complete Environmental Impact Statement (EIS) in light of the new risk analysis presented in its document. It is our belief that the government, upon reassessing the analysis used and commented on in the EIS, should have reopened public comment on the EIS. In fact, it was our understanding that the government had withdrawn its final EIS due to serious flaws in its analysis of the human health risks associated with the proposed insecticides. d

5. The public comment process should provide for public hearings on the Environmental Impact Statement. The high level of public concern related to spray programs demands greater public input in the process than has been allowed for. e

6. The document assumes that all foliage residues for the range of proposed chemicals will be the same as the level determined for carbaryl. This assumption is faulty. An adequate analysis requires that proposed chemicals be analyzed individually and on a product specific basis for not only foilage residues but for every potential exposure and environmental effect. f

7. The document throughout refers to indices of safety, such as the Average Daily Intake (ADI), which are based on a registration system at the U.S. Environmental Protection Agency that cannot at this time assess safety. The chemicals cited in the document have not been brought into compliance with modern safety standards, their files are filled with inadequate data and, as a result, related standards cannot be relied upon. g

The current process being carried out by the government in seeking public comments on its Gypsy Moth Suppression and Eradication Projects is unacceptable to us and constitutes a breach of public confidence for the reasons stated above. It is our belief that through an adequate public review and comment process important facts will be brought to light and provide for a comprehensive review of the health and environmental impact of the government's proposed spray projects. h

We appreciate the opportunity to comment.

Sincerely,

Jay Feldman
National Coordinator

State of New Jersey
DEPARTMENT OF THE PUBLIC ADVOCATE
DIVISION OF PUBLIC INTEREST ADVOCACY
CN 850
TRENTON, NEW JERSEY 08625

JOSEPH H. RODRIGUEZ
PUBLIC ADVOCATE

RICHARD E. SHAPIRO
DIRECTOR
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February 1, 1985

Gary Moorhead
Staff Officer
Plant Protection and Quarantine
Animal and Plant Health Inspection
Service
United States Department of
Agriculture
Federal Building, Room 663
6505 Belcrest Road
Hyattsville, MD 20782

Re: Draft Supplement to Gypsy Moth Suppression and Eradication Project Environmental Impact Statement, 49 Federal Register 49649 (December 21, 1984)

Dear Mr. Moorhead:

The New Jersey Department of the Public Advocate welcomes this opportunity to comment on the Draft Supplement Final Environmental Impact Statement (FEIS) for Gypsy Moth Suppression and Eradication Project, 49 Federal Register 49649 (December 21, 1984). The Department of the Public Advocate is an executive agency of New Jersey state government and is empowered to represent the public interest in administrative and court proceedings.* The Department has had a longstanding involvement in environmental issues generally and, in particular, reducing human exposure to toxic chemicals, including pesticides. The Department is currently supporting proposed legislation in New Jersey that will promote the use of alternative, non-chemical pesticide control measures, and has actively participated in meetings with the Office of Pesticide Programs of the federal Environmental Protection Agency concerning proposed farmworker protection regulations.

* N.J.S.A. 52:27E-2. "Public interest" is defined as "an interest arising from the Constitution, decisions of court, common laws or other laws of the United States or of this State inhering in the citizens of this State or a broad class of such citizens." N.J.S.A. 52:27E-30.

Our comments on the Draft Supplement FEIS primarily focus on the difficulty this document poses for adequate public review and comment on USDA policy. First, the document has been issued in a piecemeal fashion, without adequate opportunity for public comment on the FEIS as a whole. The Federal Register notice soliciting public comment on the Draft Supplement specifically limits this comment to the Supplement, thereby preventing public review of the FEIS in its entirety, including the risk assessment issued as Appendix F of the FEIS. Unfortunately, an evaluation of Appendix F, which has been re-written since public comment was solicited on the FEIS, is essential for an informed review of the merits of the Supplement. Clearly, if the public is to assess the usefulness and legal adequacy of the FEIS as a tool for evaluating the relative risks and benefits of carbaryl and alternative pest control strategies, it must be able to review the risk assessment (Appendix F) as an integral part of the entire FEIS. a

Second, the Public Advocate is concerned that the Draft Supplement is written in a manner that confuses, rather than elucidates, the issues and policy determinations. The Council on Environmental Quality (CEQ) regulations governing the writing of EIS drafts requires that they be written in "plain language . . . so that decision-makers and the public can readily understand them," and that "writers of clear prose" should be employed to insure that this goal is achieved. 40 C.F.R. §1502.8. Moreover, CEQ regulations also require that executive summaries for each EIS must "adequately and accurately summarize the statement," including the major conclusions, areas of controversy, and issues to be resolved. 40 C.F.R. §1502.12. b

Unfortunately, the Draft Supplement to the FEIS falls far short of these requirements, and consequently, fails to adequately and accurately inform the public about the gypsy moth suppression program, the health and environmental risks of a carbaryl-based program, and the available alternatives to carbaryl. The Draft Supplement is written in highly technical language that is unlikely to be understood by members of the public without advanced or specialized technical education. The Executive Summary, rather than clarify the technicalities of the Supplement, instead fails to reveal the extent of the health risks posed by carbaryl and the areas of controversy that are the subject of the Supplement itself. c

Finally, the Draft Supplement fails to provide an adequate comparative discussion of either the efficacy or health risks of carbaryl and currently available alternatives, both chemical and non-chemical based. Thus, it fails to fulfill its function as an informational document that can provide State agencies, such as the New Jersey Department of Agriculture, with sufficient comparative information about currently available gypsy moth suppression procedures to make informed decisions about which control methods to choose.

Because of the lack of clarity in the Draft Supplement, the inadequacy of the Executive Summary, and the incomplete discussion of health risks and comparative data, the Department of the Public Advocate suggests that a public hearing be held on the FEIS and Draft Supplement so that the public can be fully apprised of the major issues and controversies concerning carbaryl use and to more fully develop the necessary comparative information concerning alternative methods of gypsy moth suppression and control.

Very truly yours,

Sharon A. Treat

SHARON A. TREAT
Assistant Deputy Public Advocate

SAT/cat

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COMMONWEALTH of VIRGINIA

Council on the Environment

KEITH J. BUTTLEMAN
ADMINISTRATOR

903 NINTH STREET OFFICE BUILDING
RICHMOND 23219
804-786-4500

January 31, 1985

Mr. Gary E. Moorehead
Staff Officer
U.S. Department of Agriculture
Animal and Plant Health Inspection
Service-PPQ
Federal Building, Room 663
Hyattsville, Maryland 20782

Dear Mr. Moorehead:

The Commonwealth of Virginia has completed its review of the Draft Supplement to the Final Environmental Impact Statement on Gypsy Moth Suppression and Eradication Projects. The Council on the Environment is responsible for coordinating Virginia's review of federal environmental documents and responding to appropriate federal officials on behalf of the Commonwealth. The following agencies joined in this review:

Department of Agriculture and Consumer Services
Department of Conservation and Historic Resources
Department of Health
State Water Control Board.

The Commonwealth continues to support the preferred alternative, Integrated Pest Management (IPM), which consists of funding of a combination of measures such as biological and chemical insecticide application, inspections, application of the gypsy moth pheromone, and quarantines. Our support has one condition related to the chemical insecticide program component, which is explained below.

Laboratory toxicity tests on three of the four chemical insecticides (all but trichlorfon, for which information was not available) were reviewed by the State Water Control Board. These indicate that aquatic life could be harmed in the event diflubenzuron or carbaryl is used. Acephate used at the recommended dosage did not give rise to adverse effects on aquatic biota. Carbaryl, on the other hand, is toxic to such biota in different capacities. It readily hydrolizes to 1-naphthol in model ecosystems and in cell culture medium. The 1-naphthol is at least as toxic as carbaryl. For these reasons, carbaryl should be excluded from the IPM alternative. Diflubenzuron is not toxic to sensitive fish species used in tests (rainbow trout or coho salmon), but was somewhat toxic to invertebrates; we continue to support its inclusion in the IPM alternative.

a

Public water supply sources in Virginia are likely to be affected by spraying of chemical insecticides. Therefore, water sampling in such sources will have to be done before and after spray applications. Notification of such applications should be given to Thomas Gray of the Health Department's Bureau of Water Supply Engineering (telephone (804) 786-1768) at least 60 days before spraying is to take place. b

The worst-case analysis in Appendix F provides valuable information on the risks associated with the use of chemical insecticides. This appendix is well-written and forthright about what is known and not known. It has been helpful in our analysis of the gypsy moth suppression and eradication program. c

Additional agency comments are attached. Thank you for the opportunity to review the Draft Supplement.

Sincerely,

Keith J. Buttleman

Keith J. Buttleman

Enclosures

cc: The Honorable Betty J. Diener
Ms. LaVern Corkran, SWCB
Dr. Robert B. Stroube, DOH
Mr. Earl A. Finch, VDACS
Mr. Leon E. App, DCHR

COMMONWEALTH of VIRGINIA

STATE WATER CONTROL BOARD
2111 Hamilton Street

Richard N. Burton
Executive Director

Post Office Box 11143
Richmond, Virginia 23230-1143
(804) 257-0056

January 25, 1985

Mr. Charles H. Ellis, III
EIS Coordinator
Council on the Environment
903 Ninth Street Office Building
Richmond, Virginia 23219

RE: Draft Supplement to FEIS on Gypsy Moth Suppression & Eradication Projects

Dear Mr. Ellis:

We have reviewed the draft supplement and offer the following comments:

The Gypsy Moth Suppression and Eradication projects involve the use of four different insecticides. They are acephate, carbaryl, diflubenzuron, and trichlorfon. As stated in this document, the exposure and subsequent dose to fish are assumed to range from a realistic value of 50 ppb (.05 mg/l) to a worst case value of 707 ppb (.707 mg/l) for every 1 lb. a.i./acre applied.

After reviewing laboratory toxicity tests on three of the four insecticides, there could be adverse effects to the aquatic community if carbaryl and diflubenzuron were used. There was no information concerning aquatic biota for the insecticide trichlorfon. There were no adverse effects of aquatic biota with the use of acephate at the recommended dosage stated in this document.

In two different toxicity tests using diflubenzuron, it appeared that the insecticide was not toxic to fish, but toxic to invertebrates. The following are results of studies cited:

1. The use of diflubenzuron was not toxic to either rainbow trout or coho salmon (sensitive species) up to 150 mg/l, the maximum concentration tests for a 96-hr. period.

2. For five months at concentrations of .0001, .001, .010, and .050 mg/l, effects of a continuous exposure to diflubenzuron on a biological community in a complex laboratory stream were assessed. The insect fauna suffered direct toxic effects at concentrations of .001 mg/l and greater. The algal and fungal floras were mildly affected at the same

concentrations. No effects were observed on the bacteria, oligochaetes or gastropods at any of the test concentrations. Within the insect fauna, differences in sensitivities were observed: mayflies and stoneflies were affected at .001 mg/l, dipterans were affected at .01 mg/l, and coleopterans were unaffected at any of the test concentrations.

The use of the insecticide carbaryl is toxic to the aquatic biota in different capacities. Carbaryl readily hydrolizes to 1-naphthol in model ecosystems and in cell culture medium. The 1-naphthol has been observed to undergo no further breakdown, thus persisting in cell culture medium for at least 48 hrs.

Previous studies have shown 1-naphthol to be more toxic than its parent compound, carbaryl, to several species of mollusks and to several species of fish. A recent study has shown 1-naphthol to be as toxic as carbaryl to protozoal cultures.

In a 10-day static toxicity test, 1-naphthol was approximately 5 times more toxic than carbaryl in goldfish and in killifish, 1-naphthol was twice as toxic as carbaryl. Furthermore, all surviving fish exposed to 1-naphthol exhibited neurological trauma, whereas no neurological trauma was observed in fish exposed to carbaryl.

Based on these previous studies, it may be suggested that 1-naphthol may be responsible for a significant portion of the effects observed as a result of the application of carbaryl.

One other toxicity test using carbaryl is cited.

1. When fathead minnows were exposed to five concentrations (0.008-0.68 mg/l) of the insecticide carbaryl for months and throughout a life cycle, the highest concentration prevented reproduction and decreased survival. At the 0.68 mg/l concentration, carbaryl appeared to contribute to mortality of larvae (produced by unexposed parents) within 30 days of hatching.

   The 96-hr. median tolerance concentration (TL50) and the lethal threshold concentration (LTC) for 2-month-old fathead minnows were 9.0 mg/l.

This study demonstrates that a concentration of 0.68 mg/l carbaryl adversely affects survival and spawning of fathead minnows.

With these toxicity tests in mind, carbaryl should not be used for the suppression and eradication of gypsy moths due to its adverse effects on the aquatic environment. There are other insecticides, such as acephate, that would not pose a problem with the non-target organisms.

Sincerely,

LaVern H Corkran

LaVern H. Corkran, Program Manager
Permits
Office of Water Resources Management

:scj

xc: C. E. Easlick-EIS Coordinator
Camille Cook -OERS
OWRM Files 60-0050 and 60-0051

COMMONWEALTH of VIRGINIA

S. MASON CARBAUGH
COMMISSIONER

BILLY W. SOUTHALL
DIRECTOR

DEPARTMENT OF AGRICULTURE AND CONSUMER SERVICES
Division of Product and Industry Regulation
P. O. Box 1163, Richmond, Virginia 23209

January 25, 1985

Mr. Charlie Ellis
Council on the Environment
903 Ninth Street Office Building
Richmond, Virginia 23219

Dear Charlie:

Enclosed are comments to the Gypsy Moth Suppression and Eradication Projects which have been prepared in response to your request of January 7, 1985. We would like for them to be considered in Virginia's reply to the proposal.

Sincerely,

Donald H. Kludy
State Entomologist & Chief
Bureau of Plant Protection
and Pesticide Regulation
804/786-3515

DHK/cbf

cc: Earl Finch

8. REVIEW INSTRUCTIONS:

A) Please review the document carefully. If the proposal has been reviewed earlier (e.g., if the current document is a Final EIS), please consider previous comments.

B) Prepare your agency's comments in a form which would be acceptable for responding directly to a project sponsoring agency.

C) Use the space below for your comments. If additional space is needed, please attach extra sheets.

Return your comments to:

Charles H. Ellis III
Environmental Programs Analyst
Council on the Environment
903 Ninth Street Office Building
Richmond, Virginia 23219

Charles H. Ellis III
Environmental Programs Analyst

COMMENTS

There are water sources for public water supplies impacted by this project. The USDA, Forest Service, should contact Thomas Gray, Assistant Technical Services Chief, Bureau of Water Supply Engineering, Virginia Department of Health, 60 days prior to spraying forest land with pesticides which could affect the water sources.

Water quality sampling of the water sources will have to be done prior to the spraying and after the spraying.

Mr. Gray can be contacted at 804-786-1768.

(SIGNED) Robert Stroube M.D. (DATE) January 15, 1985

(TITLE) Assistant Commissioner, Office of Health Protection & Environmental Management

(AGENCY) State Health Department

Title of Proposal:	Gypsy Moth Suppression and Eradication Projects
Sponsor of Proposal:	USDA-Forest Service and Animal and Plant Health Inspection Service
Type of Document:	Draft Supplement to the Final Environmental Impact Statement
Commenting Agency:	Virginia Department of Agriculture and Consumer Services

The Virginia Department of Agriculture and Consumer Services (VDACS) has reviewed the subject document and makes the following comments.

The document is well written and comprehensive. The text changes clarify ambiguities present in the Final 1984 Environmental Impact Statement (FEIS). Of particular note to VDACS is the clarification of non-participation by individuals or residents in proposed suppression projects. Our experience indicates that a voluntary gypsy moth suppression program is met with less resistance from the public in general. Residents and landowners within a proposed suppression area feel they have more control and input into the program and its scope. This is particularly evident in a project involving more than one state in which the cooperating states have differing approaches to making the public aware of the voluntary nature of the program.

Appendix F, the "worst case" analysis, is also well written and comprehensive. The analysis is very detailed and provides decision makers with the probability of risks, both normal and abnormal, associated with the use of chemical insecticides. Whenever uncertainties or data gaps in the literature were

encountered, this fact was clearly identified. Upon identifying that uncertainties existed, the assumptions made were realistic, clearly stated and the situations described when applying pesticides, including worst case scenarios, were appropriate. The statistical models and equations used in the analysis were explained such that a lay person could understand and follow the analysis for intelligent decision-making.

Overall, we believe this to be a well written, comprehensive document.

Virginia Department of Agriculture
and Consumer Services
Bureau of Plant Protection
and Pesticide Regulation
January 25, 1985

19C

COMMONWEALTH of VIRGINIA

B. C. Leynes, Jr.
Director

Department of Conservation
and Historic Resources

1100 Washington Building
Capitol Square
Richmond, Virginia 23219
(804) 786-2121

January 24, 1985

MEMORANDUM

TO: Mr. Charles H. Ellis, III, COE

FROM: Leon E. App

SUBJECT: EIS #630 - Gypsy Moth Suppression and Eradication Projects

Our Division of Forestry reviewed this project and has the following comment:

We find the Draft Supplement to be a rigorous effort to address the question of public health and proposed gypsy moth suppression and eradication projects. The human health risk analysis, including the "worst case analysis" is a welcome addition to the EIS; will provide valuable information for the discussions which inevitably occur when gypsy moth control with insecticides is proposed.

ptc
cc: Mr. Phil Grimm, Division of Forestry

31 January 1985

Gary E. Moorehead, Staff Officer
USDA - APHIS - PPQ
Federal Building, Room 663
Hyattsville, Maryland 20782

RE: Draft Supplement to the Final EIS - Gypsy Moth Suppression and Eradication Projects

Dear Sir:

As part of the ever-growing number of persons who are members of what the Draft Supplement (DS) terms "sensitive populations," we are appreciative of the fact that some official recognition has at last been granted us. At the same time, the current DS remains arbitrary and insufficient in dealing with the potential for health problems that pesticides pose for people like us.

First, the new factor of 100, while better than the old one of 10, is just as arbitrary, as the document admits (F-79). Without access to comparative numbers, but as parents of children unable to drink city water contaminated by fluoride and chlorine, or even to drink untreated well water that has passed through PVC pipes, we have cause to have grave doubts about the adequacy of a factor of 100. a

Second, because the factor of 100 appears less than sufficient, and because carbaryl, for example, takes 7 to 10 days to degrade in sunlight and longer in the soil, simply leaving during the spray is insufficient protection. And as the DS admits, "consumption of food presents a dilemma because food purchased at local retail outlets could contain residues of these same insecticides which are registered for use on agricultural crops" (F-79). But the DS merely notes the dilemma while doing nothing to factor the problem into its worst case analysis, allowing it to slip away as if it conveniently did not exist. b

Third, leaving the spray area may be an extended problem for the sensitive. During the 1982 eradication program in Salem, Oregon, we were advised by a Union Carbide official, Antoine Pueche, to absent ourselves for two to three weeks after each spray, or for a total of 4-6 weeks. Thus removal poses an extraordinary burden on the sensitive populations by denying them access to their homes for extended periods without due process. A worst case analysis must consider the costs of such removals. c

Finally, the DS analysis chooses to see human health with a remarkable tunnel vision, as if mutagenicity, teratogenicity, and carcinogenicity were all that is involved in human health. Whether our sensitive children would suffer any of these effects is open to question. But they do, and have, suffered profoundly debilitating effects especially in the central nervous system, from minute exposures even to substances generally considered benign, such as floor wax. And the effects of exposures to synthetic insecticides and herbicides have inevitably been even more profound. The difference between a child classified by federal standards as talented and gifted (and d

achieving at that level) and one utterly unable to cope with the routine demands of the classroom is clearly a significant difference in health. Any DS which limits health concerns so drastically as this one does is hopelessly far from adequate.

Beyond the analysis for sensitive populations, at least two other deficiencies in the DS cry out for comment here. First, the assumption (F-49 and F-50) that a person would experience no more than either six eradication exposures or one suppression exposure per ten years seems patently absurd. In a four year period, one area of Salem was subjected to three projects or nine exposures, and state officials here now publically concede the growing possibility that Oregon might have to shift from eradication to control, leaving open the substantial possibility that some Oregonians will undergo multiple eradication and suppression projects.

And finally, the casual dismissing of synergistic effects (F-81) because naturally occurring chemicals are encountered at much higher doses is rather like saying that because apples and oranges are consumed in higher quantities, we needn't concern ourselves with the synergistic effects of DDT and 2-4-5-T.

All these deficiencies in the DS are clear even to laymen unskilled in statistical analysis and demonstrate that even the latest revision falls far short of being an adequate worst case analysis. In this document, as in all its predecessors, it appears to us that the writers have sought only to meet the bare requirements of the letter of the law and to downplay or deny the potential for human costs of spray programs.

Sincerely,

Kenneth S. Nolley

Kenneth S. Nolley

Janet G. Nolley

Janet G. Nolley
3358 Pringle Road SE
Salem, OR 97302

Glen and Elaine Olsen
Citizens for the Safe Control of Gypsy Moths
354 Hrubetz Road, SE
Salem, Oregon 97302

Feb. 1, 1985

Gary E. Moorehead, Staff Officer
USDA - APHIS - PPQ
Federal Building, Room 663
Hyattsville, Maryland 20782

Dear Mr. Moorehead:

Regarding the Draft Supplement to the Final EIS for gypsy moth spray projects, we are quite frankly disappointed by the apparent attempts to minimize rather than honestly examine the environmental and human health risks associated with the use of chemical insecticides.

The assumptions upon which this current analysis is based appear to ignore whole segments of the population. Your risk assessments address effects upon a hypothetical "average" 70 kg adult. What about the children? Does the USDA expect us to believe that the chemical dose received by a child would have the same impact as that of a 70 kg adult? Does the USDA expect us to disregard the concerns raised about the impact these chemical doses have on children? a

According to Dr. Ruth Shearer, molecular geneticist and toxicologist, "At the rate of application of carbaryl planned for Salem (10.4 mg/ft^2), the World Health Organization's ADI (acceptable daily intake) of 0.01 mg/kg/day would be reached by a 33 pound child touching 2 square inches of ground shortly after spraying. Carbaryl persistence on soil varies widely with the type of soil, but using the shortest reported half-life of eight days, the child described above could absorb his ADI from only eight square inches of ground right before the second spraying, and his ADI would be exceeded for much of the summer by normal play on the ground." What are the long term effects such repeated exposures might cause for our children? b

The credibility of this FEIS comes into question regarding the estimated number of exposures to chemical insecticides the "average" person might receive during a 70 year lifespan (see pages F-49 and F-50). It taxes the imagination to think that you who oversee countless eradication and suppression gypsy moth projects throughout the USA are unaware of the numerous repeated exposures to chemical insecticides you've subjected citizens to throughout the Northeast during annual gypsy moth spray seasons. Even in our own city of Salem, residents have been repeatedly exposed to insecticides for the past three years. Recent as well as past history certainly contradict your misleading estimates that an individual will be exposed to only two projects per lifetime resulting in six exposures in one 70 year period. c

The numerous references to comparing risks associated with chemical insecticides to risks associated with drinking diet sodas and smoking cigarettes appear to be attempts to trivialize serious concerns raised d

not only by citizens potentially impacted by the sprays, but also the many concerns raised within the scientific community. The consumption of diet sodas and the smoking of cigarettes are actions some individuals choose to take. The exposure to chemical sprays is not something presented to us as a choice, rather, chemical sprays are imposed on citizens without their permission. We personally do not choose to drink diet sodas, nor to smoke cigarettes. We do not wish to have the chemical of your choice sprayed upon us against our will.

One of the more callous instances of disregard for an entire segment of the population is your recommendation on page 23 to limit exposure to trichlorfon by using it only in sparsely populated areas. While acknowledging that "..this would not reduce exposure to individuals in the treated area," you suggest "...But it would reduce the probability of heritable mutations occurring." You state that the risk of heritable mutations "cannot be quantified" with regards to trichlorfon, yet you seem to be willing to let the people living in rural areas encumber the risks of living with a possible chemical time bomb. That reeks of a political decision which is grossly unfair and unethical. e

We, too, are concerned about the damage gypsy moths can cause to our beautiful forests. The means to halt that damage without jeopardizing human health is already available. Biological alternatives such as trapping and bacillus thuringiensis are proving to be safe and efficient. f

No tree, no forest, no lumber company is so important as to warrant risking the health of our children, the elderly, the chemically sensitive, the unborn, and our future generations. If even one baby is born with a birth defect, if even one person out of a million dies of cancer because of your chemical insecticide spray program, that is one person too many. g

Sincerely,

Elaine Olsen

Glen Olsen

Glen and Elaine Olsen

NEW JERSEY COALITION FOR ALTERNATIVES TO PESTICIDES
P.O. BOX 627
BOONTON, NEW JERSEY 07005
(201) 334-7975

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RCVD. FOSS 2-5-85

February 1, 1985

Gary E. Moorehead, Staff Officer
USDA - APHIS - PPQ
Federal Building, Room 663
Hyattsville, Maryland 20782

Re: GYPSY MOTH SUPPRESSION AND ERADICATION PROJECTS
DRAFT SUPPLEMENT TO THE FINAL EIS

New Jersey Coalition for Alternatives to Pesticides (NJCAP) has the following comments concerning certain pages of the Draft Supplement.

PAGE F-18 OF DRAFT SUPPLEMENT:

"Schulze (1979) reported finding no detectable exposure to residents in the spray area."

NJCAP COMMENTS:

This statement definitely needs clarification. According to Schulze's, et al. report entitled "Assessment of Human Exposure Following Aerial Applications of Carbaryl for Gypsy Moth Suppression in New Jersey: 1978 and 1979 studies", air samples as well as urine samples from people were collected and analyzed. If the above statement in the Supplement refers to exposure to residents based on urine sampling then the statement in the Supplement is incorrect. Alpha-naphthol, one of the break-down products of carbaryl, served as evidence that people were exposed to carbaryl. Photocopies of pages 21 and 18 from Schulze's report are provided as rebuttal to the Supplement's statement that no detectable exposure to residents in the spray area occurred. If the statement in the Supplement refers to air sampling, then it should be qualified with the statement appearing on page 15 of Schulze's report which is attached.

a

density was defined as essentially open or devoid of trees, moderate tree density represented an area covered approximately 50% by the forest crown cover, while the heavy tree density referred to virtually total coverage of the area by the forest crown cover.

RESULTS AND DISCUSSION

Air Monitoring

Carbaryl was not detected in any of the 1978 or 1979 air samples. Due to a potential analytical problem within the laboratory conducting the 1978 analyses, however, the data were judged to be of questionable reliability.

The failure to detect carbaryl in air during the 1979 Study may have resulted from a variety of factors. From the TLC plate sampling, little of the applied carbaryl reached ground level, particularly as the forest crown density increased. Further, all spray droplets which reached the ground did so within 15 minutes. As such, the lower amounts of carbaryl available at ground level and the short time in which the spray particles remained dispersed in air greatly decreased the likelihood of detection.

Secondly, carbaryl in oil formulations is unlikely to volatilize rapidly, particularly in early morning temperatures when applications are generally conducted. The low volatility of the formulation would result in air concentrations of carbaryl sufficiently low to avoid detection during the 4-hour sampling period.

Finally, the low volume of air sampled ($\bar{x}$ = 6.68 liters/hour or 26.72 liters for the 4-hour sample) may have been insufficient to provide detectable levels of carbaryl at concentrations present at the time. Further, there is little published data on the capture efficiency of florisil for cabaryl, although preliminary experimentation indicates that florisil may be effective.

Urine Data - 1978

None of the 138 urine samples showed the presence of blood in the urine, as tested by the dipstick method. Three samples tested positive for urinary glucose:

total number of urine samples (n = 78) tested by the dipstick method, none were positive for urinary blood, glucose, or protein.

A between-group comparison of pre- and during exposure α-naphthol data (Table 1) suggests a correlation between excretion of the metabolite and the anticipated level of exposure for each group. The individuals from Group 1 (n = 2), who were known to be involved in a number of carbaryl applications during the day of monitoring, showed a during exposure mean ($\bar{x}$) of 91.55 µg α-naphthol/g creatinine. These individuals had not been exposed to carbaryl for at least 8 days prior to the test application. Jndividuals from Group 2 (n = 4) who conducted the study and, hence, were directly exposed to a single application only, exhibited a $\bar{x}$ of 31.5 µg α-naphthol/g creatinine. All during exposure urine samples from Groups 1 and 2 were positive. With one exception, all pre-exposure samples were negative; the single positive from Group 2 occurred in an individual with known exposure to carbaryl from home garden use. All pre-exposure samples from Group 3 were negative for α-naphthol, while 9 of 27 during exposure samples were positive. As a whole, Group 3 showed a $\bar{x}$ of 6.30 µg α-naphthol/g creatinine; the $\bar{x}$ of only those individuals exhibiting positive values was 18.9 µg α-naphthol/g creatinine. These data indicate a trend similar to that found in the 1978 Study: occupationally exposed individuals showed a consistently higher level of α-naphthol excretion than those who are incidentally exposed.

There were, however, certain application differences between the 1979 Study as compared to the 1978 Study. In the 1979 Study, the application occurred at 10:30 a.m., when 85% of the residents were not present at the site. Of the four people that remained, only one had a positive during exposure urine. The remaining eight individuals who donated positive during exposure urines obviously became exposed after returning to the site. In the previous study, the application occurred at 7:00 a.m. while all 26 participants were at home; yet only one exhibited a during exposure urine specimen positive for α-naphthol. The 1978 cohort could have been expected to show greater exposure since they would have had greater opportunity to contact carbaryl-contaminated surfaces while going to, as well as returning from routine daily activities. As the application rate (0.75 lbs. carbaryl/acre), commercial product (Sevin-4-Oil), and method of application were identical in both years, presumably some extrinsic factor(s) accounted for this apparent disparity. On recall, it seemed that the forest crown cover was more uniformly dense at the 1978 study site which would imply that less carbaryl would be expected to reach the ground. Secondly, the 1979 cohort appeared to be more active in the community. This would tend to

on at least one individual with the left shoulder, right shoulder, right forearm, and chest patches being contaminated on two individuals. While the purpose of this monitoring was to assess direct exposure from the spray particles, it is impossible to determine whether the levels found are fully the result of such exposure or are, in part, the result of secondary exposure from contact with contaminated vegetation or objects at each site. The amount of carbaryl on all uncovered patches for each individual ranged from 10-20 µg/150 cm^2 with a $\bar{x}$ of 15 µg carbaryl/person. No correlation between carbaryl levels found and tree density was apparent.

SUMMARY AND CONCLUSION

The results of both the 1978 and the 1979 studies indicate a positive association between carbaryl exposure and urinary excretion of α-naphthol. In both studies, the most highly and frequently exposed groups excreted the highest levels of α-naphthol. For Group 2 (1978), exposure was demonstrated by excretion of α-naphthol in seven of nine individuals within 24 hours after exposure (during sample), and the total lack of the metabolite in the pre-exposure sample. This indicated that exposure to carbaryl can be documented in individuals within a target area despite an inability in this study to demonstrate the presence of the insecticide in the air. In Group 2 (1979), individuals occupationally exposed to a single application also consistently excreted the metabolite following exposure, but at lower levels than Group 1 (1979) who received multiple exposures. Area residents (Groups 3) in both years exhibited the lowest α-naphthol levels and the lowest frequency of positive readings. In the 1978 Study, only 4 of 26 residents showed positive urine readings; in the 1979 Study, 9 of 27 (33%) showed positive readings, but this was still the lowest level and frequency of exposure. The majority of Group 3 individuals (1979) appear to have become exposed to carbaryl via secondary contact with contaminated objects or vegetation, since only one of the nine participants showing positive readings was on site at the time of application. Further, from the serial sampling of Group 2 (1979) individuals, 12-hour (night) urines provided only a partial picture of total carbaryl metabolism. α-Naphthol was found to be excreted within 6 hours of exposure with measurable levels of the metabolite continuing for at least 36 hours. The frequency of individuals with positive α-naphthol urines was highest in sparse tree density residences (42%) and lowest in heavily wooded residences (20%).

NJCAP's Comments on the Draft Supplement

Page F-22 and 23 of Draft Supplement:

"Studies of residues on vegetable crops or grass illustrate that initial residues of insecticides range from 1 to 100 ppm depending on the insecticide and type of the vegetation..." "These residues degrade to nondetectable levels within 10 to 14 days on vegetation except for grass which can have detectable residues for up to 28 days..."

NJCAP Comments:

Perhaps the following studies should be mentioned in the Supplement. According to Devine (1971) in USDA 1974 Final EIS on Cooperative Gypsy Moth Suppression and Regulatory Program, page 105, two studies using 1 lb./acre of carbaryl showed carbaryl residues in leaves approximately 80 days after application. According to Fairchild (1970) in 1974 FINAL EIS, page 80, studies using 1 lb./acre of Sevin on maple trees in Michigan showed residues of 43 ppm after 35 days.

Page F-26 of Draft Supplement:

The low octanol/water partition coefficients,..., and 240 for carbaryl, indicate that these insecticides should not be fat soluble or accumulate in fish tissue. b

NJCAP Comments:

According to EPA's Office of Pesticide Programs report entitled "Guidance for the Reregistration of Pesticide Products Containing Carbaryl as the Active Ingredient" 056801 dated March 30, 1984, "preliminary data indicate that there may be a potential for carbaryl to accumulate in catfish, crayfish, snails, duckweed and algae", page 17. c

Page F-30 of Draft Supplement:

"Kuhr and Dorough (1976) report on a number of studies involving the biostability of carbaryl on crops. The spinach group of crops had initial residues of 52 ppm that degraded to about 9 ppm in one week. Lettuce residues were usually lower and dissipated shortly after application partly because of dilution by growth." "Kuhr and Dorough (1976) also reported that simple washing shortly after spraying removed more than 90% of carbaryl residues. Therefore, vegetables were assumed to have an initial range of residues from 1 to 5 ppm after washing and these residues degraded to zero (undetectable) within 14 days."

NJCAP Comments:

According to Organic Gardening, October 1984, "The Trouble with Carbaryl" by Warren Schultz Jr., d

NJCAP's Comments on the Draft Supplement

> Researchers in India sprayed a tomato crop with a 0.2% carbaryl solution, then harvested the fruit at intervals and checked for residues. It took 25 days for the substance to become completely dissipated. Residues in the fruit picked 5 days after spraying exceeded the maximum tolerance level of five parts per million as set by the World Health Organization. Even washing the fruit didn't remove all the pesticides. Tomatoes that contained 10.7 ppm carbaryl on the day of spraying were washed for 2 minutes. After that the residues in the fruits still measured 4.06 ppm. The other study showed similar results on cauliflower. The crop was sprayed with 2.5 kg. of Sevin 50 wettable powder per hectare, a common rate. The initial deposit of carbaryl on the curds was 16.75 ppm. It took 6 days for the residues to dissipate below the accepted 5 ppm tolerance level. After 15 days there was still a detectable residue on unwashed curds. (pages 62 and 63)

It is important to note here that garden crops such as lettuce, radishes, spinach and asparagus are harvested as early as the middle of May in New Jersey, the time in which spraying for gypsy moths is conducted. There is a formulation of Sevin intended for garden crops - Sevin Sprayable - and the harvesting schedule states that spinach and leaf lettuce must not be harvesting until 14 days after application, head lettuce and radishes 3 days afterwards and asparagus one day afterwards (see attached). Sevin 4 Oil used in gypsy moth control is <u>not</u> intended for use in home gardens so what harvesting schedule applies if Sevin 4 Oil falls on the crops in the garden.

NJCAP's Comments on Draft Supplement

Page F-49 of Draft Supplement:

An exposure in a lifetime includes: exposure from the direct application, secondary exposure or contact with spray residues on grass, foliage, cars, etc. and eating or drinking contaminated foods.

NJCAP Comments:

Consideration must be given to the fact that there will be additional exposure to gypsy moth sprays from neighbors spraying carbaryl or other chemicals for gypsy moths because they wish to have applications besides the one aerial application by the state. Other exposures result from neighbors using these chemicals in their gardens and/or for agricultural use. One must not only consider the numbers of exposures from gypsy moth sprays but should also consider the immense amount of pesticides people are exposed to from indoor spraying of schools, hospitals, offices, etc. as well as insect control by neighbors (insects other than gypsy moths) and pesticideexposure from agricultural use.

Dr. Ruth Shearer, a molecular geneticist, is alarmed about the widespread use of carbaryl, especially aerial sprayings aimed against gypsy moths.

> She believes that a significant danger to children comes from dermal exposure. Carbaryl is rapidly and nearly completely absorbed through human skin, she warns. Using World Health Organization figures, she calculated that children are endangered whenever carbaryl is sprayed over wide areas. The WHO has set an Acceptable Daily Intake (ADI) of carbaryl for humans at 0.01 milligrams per kilogram -- that equals 10 billonths of our body weight. The intake can be oral, dermal or respiratory. For example, she says, a proposed aerial spraying of one pound of Sevin per acre (that's 10.4 milligrams per square foot) would mean that a 33-pound child would exceed his ADI if he touched more than 2 square inches of soil, table, bench or foliage in the spray area on the day of the spraying. Even a week later he could safely touch only 4 square inches per day.
>
> From Organic Gardening, October 1984
> "The Trouble with Carbaryl" by
> W. Schultz Jr.

NJCAP's Comments on the Draft Supplement

Page F-71 and F-80 of Draft Supplement:

Reference is made to a New Jersey Department of Health study in Cape May County, NJ. The Draft Supplement stated that the study concluded that there was no increase in birth defects in the counties where carbaryl was used, compared to counties where it wasn't.

NJCAP's Comments:

Testimony provided by the New Jersey Department of Health before the Governor's Science Advisory Committee (special hearing on gypsy moths) at State House, Trenton, NJ, Feb. 16, 1982 stated that this study was at best inconclusive due to the fact that there was not enough time to do a proper epidemiologic study. We would appreciate it if this fact was made known in future impact statements when reference is made to this study. f

Page F-75 of Draft Supplement:

In all cases, the lifetime risks of cancer resulting from exposure to carbaryl or trichlorfon used to control gypsy moths are lower than the risk of cancer from smoking 2 cigarettes, drinking 40 diet sodas, or having a single X-ray in a lifetime, which are all in the order of 10^{-6}, or one in a million risk.

NJCAP's Comments:

Smoking cigarettes, drinking diet soda and having X-rays are individual decisions and the cancer risks associated with these decisions are risks the individual chooses to take not risks that are forced upon him or her. An aerial spraying programs exposes individuals to chemicals that he may not wish to be exposed to. In New Jersey, the gypsy moth aerial spraying program finds the individuals with less chance as to whether or not they wish to be exposed. This must be taken into consideration in any risk assessment conducted. g

Page F-77 of Draft Supplement:

Ames (1983 and 1984) pointed out that humans are continually exposed to natural chemicals in our diets which have tested positive in various in-vitro mutagenicity tests. He concluded that exposure to these chemicals "is likely to be several grams per day -- probably at least 10,000 times higher than the dietary intake of man-made pesticides." If a mutation occurred, it would be impossible to determine whether it was caused from pesticide exposure or exposure to one or many of the natural mutagens known to exist in our environment.

NJCAP's Comments:

Ames points out in his article entitled "Dietary Carcinogens and Anticarcinogens", (Science, September 1983, Vol. 221) the human diet contains a great variety of natural mutagens and carcinogens as well as many natural antimutagens and anti-carcinogens. It is our opinion that having evolved with these foods that perhaps man has adapted mechanisms for dealing h

NJCAP's Comments on the Draft Supplement

with these naturally occurring carcinogens and mutagens. However, Ames does point out in his article that many of these plant toxins may be "new" to humans in the sense that the human diet has changed drastically with historic times. Thus we are of the opinion that every effort we make to reduce exposure to carcinogens and mutagens whether they are from pesticides or naturally occuring in food will lower the total load we receive and is a step in the right direction. It should be pointed out that these chemical pesticides are unnatural substances with which the body has not had the time or ability to develop physiological mechanisms in order to cope with them.

Page F-6 in Draft Supplement:

In order to establish safe levels of pesticides the NOEL is divided by a safety factor.

NJCAP's Comments:

Once the NOEL is divided by the safety factor, the result is multiplied by 60 to get a dose for the average person. The EPA has decided that the average person weighs 60 kilograms or 132 pounds. EPA has even decided on one "average" diet and has calculated what percentage each food represents. But who eats this average diet and weighs exactly 132 pounds? If weight is taken into account, children may eat enough of some foods to exceed the EPA's safe dose of a pesticide. And young children are more sensitive to toxic chemicals than adults. One must also taken into consideration the fact that some crops exceed pesticide tolerance levels established by EPA.

IN SUMMARY, NJCAP questions the qualifications of those who prepared the risk assessment. In addition, we belive people should not have to accept unknown risks in order to control an insect considered a "public nuisance" and not a public health hazard. In New Jersey, there is not an adequate plan for identifying and keeping statistical records on pesticide illnesses. Physicians often do not recognize the symptoms of pesticide illness, and there is no law which requires physicians who suspect pesticide poisoning to submit blood and/or urine samples to be analyzed. Thus, many cases of actual and/or suspected pesticide poisonings are unreported. Since US Department of Agriculture along with the individual states are conducting gypsy moth spray programs, we strongly recommend and feel it to be your responsibility to set up a program to alert physicians and veterinarians concerning the syptoms, diagnosis, treatment and reporting of pesticide illnesses associated with your program.

We appreciate this opportunity to comment.

Sincerely,

Susan Shaw

Susan Shaw

Sevin® Sprayable

UNION CARBIDE

Carbaryl Insecticide

For control of insect pests.

CAUTION: Keep out of reach of children.
For agricultural or commercial use only.

Active Ingredient:
Carbaryl (1-naphthyl methylcarbamate), 80% by wt.

Inert Ingredients: . . . . . 20% by wt.

IN CASE OF EMERGENCY PHONE COLLECT (24 HOURS A DAY) IN U.S.A. (304) 744-3487

GENERAL INFORMATION

SEVIN SPRAYABLE is a dry powder for dispersion in water and application as an insecticidal spray in hydraulic sprayers, mist blowers, low-gallonage ground equipment and aircraft. The directions on this label are based on tests and field experience relating to (a) effectiveness, (b) possible injury to plants and animals, and (c) residues in food, feed and milk. READ THIS LABEL BEFORE USE. STRICTLY OBSERVE LABEL DIRECTIONS AND CAUTIONS, AND APPLICABLE FEDERAL AND STATE REGULATIONS.

Treated areas may be reentered immediately after the spray has dried.

PREHARVEST AND GRAZING USE INFORMATION AND LIMITATIONS

Tolerances established under the Federal Food, Drug and Cosmetic Act permit the sale of crops bearing probable carbaryl residues when this product is used in accordance with label directions. Do not plant rotational crops not listed on this product label within 18 months following treatment. If used as directed, treated forage may be grazed or used as feed for dairy and meat animals without causing illegal residues in meat or milk. This product may be applied up to and including the day of harvest or grazing of forage crops. Application may be made without removing livestock from area being treated.

PLANT RESPONSE PRECAUTIONS

To avoid possible injury to tender foliage, do not apply to wet foliage or when rain or high humidity is expected during the next two days.

SEVIN injures Boston ivy, Virginia creeper and maidenhair fern. During early season, it may also injure Virginia and sand pines.

Observe label instructions on apple thinning and on combinations with certain herbicides on rice and soybeans.

DIRECTIONS FOR USE

It is a violation of Federal law to use this product in a manner inconsistent with its labeling.

Read Entire Label. Use Strictly According to Label Directions and Cautions. Do not use application methods, dosages, concentrations, or frequencies not listed on labeling. Do not apply against target pests or crops not listed on labeling. Do not mix with fertilizers.

SPRAY PREPARATION

TO ASSURE A UNIFORM SUSPENSION, AGITATE, STIR OR RECIRCULATE ALL SEVIN SPRAYABLE CONTAINERS PRIOR TO AND DURING USE. Remove oil, rust, scale, pesticide residues and other foreign matter from mix tanks and entire spray system. Flush with clean water.

Fill spray or mix tank with ½ to ¾ the desired amount of water. Start mechanical or hydraulic agitation. Slowly add the required amount of SEVIN SPRAYABLE, and then the remaining volume of water. Prepare only as much spray mixture as can be applied on the day of mixing.

MAINTAIN CONTINUOUS AGITATION DURING MIXING AND APPLICATION TO ASSURE A UNIFORM SUSPENSION. DO NOT STORE SPRAY MIXTURES FOR PROLONGED PERIODS.

COMPATIBILITY

SEVIN SPRAYABLE has been used without plant injury with most common insecticides, miticides, fungicides, nutrients, adjuvants and petroleum oil as used on citrus. If compatibility of SEVIN SPRAYABLE with another product and the resulting crop response is unknown, it should be tested on a small scale. Curdling, precipitation, greasing, layer formation or increased viscosity are symptoms of incompatibility. WHEN PREPARING COMBINATION SPRAYS, FIRST ADD SEVIN SPRAYABLE TO AT LEAST ½ THE DESIRED VOLUME OF WATER, MIX THOROUGHLY, AND THEN ADD COMBINATION PRODUCTS TO THE MIXTURE, AND THEN THE REMAINING VOLUME OF WATER. DO NOT APPLY TANK MIX COMBINATIONS UNLESS YOUR PREVIOUS EXPERIENCE INDICATES THE MIXTURE IS EFFECTIVE AND WILL NOT RESULT IN APPLICATION PROBLEMS, EXCESSIVE RESIDUES OR PLANT INJURY.

Unstable under highly alkaline conditions. Not effective if used with alkaline materials such as Bordeaux, lime-sulfur and casein-lime spreaders.

APPLICATION

On all crops, use sufficient gallonage to obtain thorough and uniform coverage.

Calibrate spray equipment to deliver the required volume.

Use 50 mesh slotted strainers in sprayers.

Avoid applications just before rainfall as poor insect control may result.

To clean the sprayer after use, drain and flush with water.

STORAGE AND DISPOSAL

Store unused SEVIN SPRAYABLE in original container only, in cool, dry area out of reach of children and animals. Do not store in areas where temperatures frequently exceed 100 °F.

Do not contaminate water, food or feed by storage or disposal.

Pesticide, spray mixture or rinsate that cannot be used according to label instructions must be disposed of according to Federal, State or local procedures under the Resource Conservation and Recovery Act.

Decontaminate empty bulk tanks and drums with triple water rinse. Do not reuse empty plastic drums or drum liners. Recondition metal drums before reuse. Dispose of in a sanitary landfill or by other approved state and local procedures.

Consult Federal, State or local disposal authorities for approved alternative procedures.

INSECT CONTROL

Apply when insects or their damage appear. To maintain control repeat at 7 to 14 day intervals or as necessary unless a shorter interval is specified below. Where a dosage range is indicated, use lower rate on young plants and early instars and higher rate on mature plants, advanced instars and adults. Thorough and uniform spray coverage is essential for effective control.

SEVIN SPRAYABLE does not control spider mites. If spider mites are a problem, use a registered miticide.

FORAGE, FIELD AND VEGETABLE CROPS

Use at least 1 gallon of finished spray per acre for aerial application and at least 3 gallons of finished spray per acre for concentrate ground application. To prepare small volumes of spray, use 1¼ tablespoonfuls of SEVIN SPRAYABLE per gallon of water where rates of 1¼ pounds per acre or 1¼ pounds per 100 gallons are indicated in the tables below.

CROP	INSECT	POUNDS OF "SEVIN" SPRAYABLE/ACRE	PRE-HARVEST INTERVAL (DAYS)	SPECIFIC DIRECTIONS
All Forage, Field and Vegetable Crops	Grasshoppers	⅔ to 1¼	See specific Forage, Field or Vegetable Crop	Use ⅔ to 1¼ pounds for nymphs on small plants or sparse vegetation in wasteland, rangeland, ditch banks and borders. Use 1¼ to 1¼ pounds for adult grasshoppers or applications to dense vegetation.
Alfalfa* Clovers	Blister beetles Mexican bean beetle	⅔ to 1¼	0	Observe plant response precautions.
	Alfalfa caterpillar Bean leaf beetle Cucumber beetles Green cloverworm Japanese beetle Leafhoppers Three cornered alfalfa hopper Thrips Velvetbean caterpillar	1¼		
	Alfalfa weevil larvae Armyworm Cloverhead weevil Corn earworm Cutworms Egyptian alfalfa weevil larvae Essex skipper European alfalfa beetle Fall armyworm Lygus bugs Stink bugs Webworms Yellowstriped armyworm	1¼ to 1¼		For alfalfa weevil larvae, if pretreatment damage is extensive, cut alfalfa and treat the stubble. Use higher rate in areas east of the Rocky Mountains. On dense growth use 25 to 40 gallons per acre with ground equipment to ensure adequate coverage.
*... For application to Alfalfa via Center Pivot Irrigation Systems, refer to "Directions for Use Through Center Pivot Irrigation Systems."				
Asparagus	Asparagus beetle	1¼ to 2½	1	
	Apache cicada Asparagus beetle	2½ to 5	Post harvest application only	Treat ferns or brush growth. Do not treat more than once every 3 days.
Beans (including blackeyed peas, cowpeas, crowder or southern peas, dry beans, green beans, lima beans, navy beans and snap beans)	Blister beetles Mexican bean beetle	⅔ to 1¼	0	Observe plant response precautions.
	Alfalfa caterpillar Bean leaf beetle Cucumber beetles Flea beetles Green cloverworm Japanese beetle Leafhoppers three cornered alfalfa hopper Thrips Velvetbean caterpillar Western bean cutworm	1¼		
	Armyworm Corn earworm Cutworms European corn borer Fall armyworm Stink bugs Tarnished plant bug Webworms	1¼ to 1⅞		
	Cowpea curculio	2½		
	Corn earworm Limabean pod borer Lygus bugs Stink bugs	2½	0	CALIFORNIA ONLY
Cabbage Broccoli Brussels Sprouts Cauliflower Kohlrabi	Flea beetles Harlequin bug	⅔ to 1¼	3	
	Armyworm Corn earworm Fall armyworm Imported cabbageworm	1¼ to 2½		
Chinese cabbage Collards Hanover salad Horseradish Kale Mustard greens Radishes Rutabagas Turnips	Flea beetles Harlequin bug Leafhoppers	⅔ to 1¼	3 (Horseradish, radishes, rutabagas and turnip roots) 14 (Chinese cabbage, collards, Hanover salad, kale, mustard greens, and turnip tops)	
	Aster leafhopper	1¼ to 1⅞		
	Armyworm Corn earworm Fall armyworm Imported cabbageworm Stink bugs Tarnished plant bug	1¼ to 2½		

CROP	INSECT		POUNDS OF "SEVIN" SPRAYABLE/ACRE	PRE-HARVEST INTERVAL (DAYS)	SPECIFIC DIRECTIONS
Carrots Parsnips Parsley	Flea beetles	Leafhoppers	⅔ to 1¼	0 (carrots) 3 (parsnips) 14 (parsley)	Treat on a 5 to 7 day schedule.
	Aster leafhopper Lygus bugs	Spittlebugs	1¼ to 1⅞		
	Armyworm Corn earworm Fall armyworm	Stink bugs Tarnished plant bug	1¼ to 2½		
Corn* (field, sweet, pop)	Armyworm Corn earworm Corn rootworm adults European corn borer Fall armyworm Flea beetles	Japanese beetle Sap beetles Southwestern corn borer Leafhoppers	1¼ to 2½	0	OBSERVE BEE CAUTION. For insects attacking silks and ears apply at 1 to 6 day intervals starting when first silks appear and continuing until silks begin to dry. For larvae in whorl and foliage feeders, apply as necessary. Optimum timing and good coverage are essential for effective control.
	Western bean cutworm		2½	0	Treat when infestation averages 15% and at 90% to 100% tassel emergence. Treatment after 100% silk emergence will reduce effectiveness
	Cutworms		2½ to 8	0	Apply in a 12 inch band, using ¼ pound (4 ounces) per 1000 linear feet of row, in at least 15 gallons of water per acre. For broadcast application use up to 8 pounds in at least 20 gallons (ground) or 5 gallons (air) of water per acre.
*. . . For application to Corn via Center Pivot Irrigation Systems, refer to "Directions for Use Through Center Pivot Irrigation Systems."					
Cotton	Cotton fleahopper Cotton leafworm Flea beetles	Striped blister beetle Thrips	⅔ to 1¼	0	Early season insect control.
	Boll weevil Bollworms Cotton leafperforator Fall armyworm Leafrollers	Leafhoppers Tarnished plant bug Yellowstriped armyworm (cotton cutworm)	1¼ to 2½	0	Treat on a 5 to 7 day schedule for as long as control is necessary. Mid and late season insect control. May be applied after bolls open.
	Lygus bugs		1¼ to 2½	0	For light to moderate populations in Western irrigated cotton.
	Pink bollworm		1⅞ to 3		Aphid populations will be suppressed by repeated applications of this insecticide.
	Stink bugs	Saltmarsh caterpillar	2½	0	
Cucumber Melons Pumpkins Squash	Pickleworm	Melonworm	⅔ to 1¼		Observe plant response precautions.
	Cucumber beetles Flea beetles	Leafhoppers Squash bugs	1¼	0	Avoid excessive applications.
Dandelion Endive (Escarole) Lettuce Salsify	Flea beetles Harlequin bug	Leafhoppers	⅔ to 1¼	3 (head lettuce & salsify roots) 14 (dandelion, endive (escarole) leaf lettuce & salsify tops)	Observe plant response precautions. Treat on a 5 to 7 day schedule after heads begin to form.
	Aster leafhopper Lygus bugs	Spittlebugs	1¼ to 1⅞		
	Armyworm Corn earworm Fall armyworm	Imported cabbageworm Stink bugs Tarnished plant bug	1¼ to 2½		
Forage Grasses Pasture	Armyworm Black grass bugs Chinch bugs Essex skipper Fall armyworm	Range caterpillars Range crane fly Striped grass looper Thrips	1¼ to 1⅞	0	To control thrips in grasses grown for seed use high spray pressure to improve penetration into boot.
	White grubs (green June beetle)		1⅞ to 2½		
Garden beet Spinach Swiss chard	Flea beetles Harlequin bug	Leafhoppers	⅔ to 1¼	3 (garden beet roots) 14 (garden beet tops, spinach, Swiss chard)	Treat on a 5 to 7 day schedule.
	Aster leafhopper		1¼ to 1⅞		
	Armyworm Corn earworm Fall armyworm	Stink bugs Tarnished plant bug	1¼ to 2½		

PRECAUTIONARY STATEMENTS
HAZARDS TO HUMANS AND DOMESTIC ANIMALS:
HARMFUL IF INHALED OR SWALLOWED. Avoid Breathing of Dust or Spray. Do Not Take Internally. Avoid Contact with Skin and Eyes.

Wear regular long-sleeved work clothing. Change to clean clothing daily. Wash hands and face before eating. Wash thoroughly after handling..

STATEMENT OF PRACTICAL TREATMENT: IF SWALLOWED, induce vomiting and seek medical attention immediately.

IF IN EYES OR ON SKIN, flush eyes thoroughly with water: wash skin thoroughly with soap and water.
NOTE FOR PHYSICIAN: Carbaryl is a moderate, reversible cholinesterase inhibitor. Atropine is antidotal. Do Not Use 2-PAM, opiates, or cholinesterase inhibiting drugs.

ENVIRONMENTAL HAZARDS:
Avoid direct application to lakes, streams and ponds. Do not apply when weather conditions favor drift from area treated. Do not contaminate water, food, or feed by cleaning equipment or disposal of wastes.

BEE CAUTION: MAY KILL HONEYBEES IN SUBSTANTIAL NUMBERS. This product is Highly Toxic to Bees Exposed to Direct Treatment or Residues on Crops. Protective Information May Be Obtained from Your Cooperative Agricultural Extension Service.

Do Not Use When Value of Bees as Pollinators is More Important than Insect Control. Before Applying, Warn Beekeepers to Locate Hives Beyond Bee Flight Range Until 1 Week After Application or to Take Equally Effective Precautions.

SEVIN is the registered trademark of Union Carbide Corporation for carbaryl insecticide.

LIMITED WARRANTY AND DISCLAIMER

1. The manufacturer warrants (a) that this product conforms to the chemical description on the label: (b) that this product is reasonably fit for the purposes set forth in the directions for use when it is used in accordance with such directions, and (c) that the directions, warnings and other statements on this label are based upon responsible experts' evaluation of reasonable tests of effectiveness, of toxicity to laboratory animals and to plants, and of residues on food crops, and upon reports of field experience. Tests have not been made on all varieties or in all states or under all conditions. THE MANUFACTURER NEITHER MAKES NOR INTENDS, NOR DOES IT AUTHORIZE ANY AGENT OR REPRESENTATIVE TO MAKE, ANY OTHER WARRANTIES, EXPRESS OR IMPLIED, AND IT EXPRESSLY EXCLUDES AND DISCLAIMS ALL IMPLIED WARRANTIES OF MERCHANTABILITY OR FITNESS FOR A PARTICULAR PURPOSE.
2. This warranty does not extend to, and the Buyer shall be solely responsible for, any and all loss or damage which results from the use of this product in any manner which is inconsistent with the label directions, warnings or cautions.
3. BUYER'S EXCLUSIVE REMEDY AND MANUFACTURER'S OR SELLER'S EXCLUSIVE LIABILITY FOR ANY AND ALL CLAIMS, LOSSES, DAMAGES, OR INJURIES RESULTING FROM THE USE OR HANDLING OF THIS PRODUCT, WHETHER OR NOT BASED IN CONTRACT, NEGLIGENCE, STRICT LIABILITY IN TORT OR OTHERWISE, SHALL BE LIMITED, AT THE MANUFACTURER'S OPTION, TO REPLACEMENT OF, OR THE REPAYMENT OF THE PURCHASE PRICE FOR, THE QUANTITY OF PRODUCT WITH RESPECT TO WHICH DAMAGES ARE CLAIMED. IN NO EVENT SHALL MANUFACTURER OR SELLER BE LIABLE FOR SPECIAL, INDIRECT OR CONSEQUENTIAL DAMAGES RESULTING FROM THE USE OR HANDLING OF THIS PRODUCT.

THIS SPECIMEN LABEL IS INTENDED TO BE USED AS A GUIDE IN PROVIDING INFORMATION ON THE GENERAL DIRECTIONS AND CAUTIONS ON THE USE OF "SEVIN" CARBARYL INSECTICIDE. ALWAYS READ THE LABEL ON THE PACKAGE BEFORE USING THE PRODUCT.

UNION CARBIDE
UNION CARBIDE AGRICULTURAL PRODUCTS COMPANY, INC.
RESEARCH TRIANGLE PARK, NC 27709

EPA Reg. No. 264-316
EPA Est. No. 10352-GA-01

Form No. AG82010-10M-TCG-11/81

Printed in U.S.A.

Pennsylvania Intergovernmental Council

P. O. BOX 11880 • HARRISBURG, PA. 17108-1880 • (717) 783-3700

February 4, 1985

Gary E. Moorehead, Staff Officer
USDA-APHIS-PPQ
Federal Building, Room 663
Hyattsville, Maryland 20782

Re: Gypsy Moth Suppression and
Eradication Projects

Dear Mr. Moorehead:

Pennsylvania's Single Point of Contact under Executive Order 12372 (Intergovernmental Review of Federal Programs) has received copies of the Draft Supplement to the Final Environmental Impact Statement for Gypsy Moth Suppression and Eradication Projects. We distributed copies to several of our reviewing agencies; these agencies do not wish to comment on the Supplement at this time.

We appreciate the opportunity to review this document.

Sincerely,

Barbara J. Gontz
Project Coordinator
Intergovernmental Review Process

BJG/abs

OFFICE OF THE SECRETARY
RESOURCES BUILDING
1416 NINTH STREET
95814
(916) 445-5656

Department of Conservation
Department of Fish and Game
Department of Navigation and Ocean Development
Department of Parks and Recreation
Department of Water Resources

GEORGE DEUKMEJIAN
GOVERNOR OF
CALIFORNIA

Air Resources Board
Colorado River Board
San Francisco Bay Conservation and Development Commission
Solid Waste Management Board
State Lands Commission
State Reclamation Board
State Water Resources Control Board
Regional Water Quality Control Boards
Energy Resources Conservation and Development Commission

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THE RESOURCES AGENCY OF CALIFORNIA

SACRAMENTO, CALIFORNIA

Mr. Gary E. Moorehead
U.S. Forest Service
Federal Building, Room 663
Hyattsville, MD 20782

January 31, 1985

Dear Mr. Moorehead:

The State has reviewed the draft supplement to the final EIS, Gypsy Moth Suppression and Eradication Project, submitted to the Office of Planning and Research.

Review of this document was coordinated with the Coastal and San Francisco Bay Commissions, Air Resources and Water Resources Control Boards, and Departments of Conservation, Fish and Game, Forestry, Parks and Recreation, Food and Agriculture, and Health Services.

We have received no comments from any of the above-mentioned entities. Therefore, the State will have no comment on this project at this time. The Department of Fish and Game has informed us that it may have comments to offer at a later time, and we will forward those to you as soon as they may be received.

Sincerely,

Charles L. Felton

for Gordon F. Snow, Ph.D
Assistant Secretary for Resources

cc: Office of Planning and Research
1400 Tenth Street
Sacramento, CA 95814

(SCH 84012305)

DEPARTMENT OF HEALTH & HUMAN SERVICES

Public Health Service

Centers for Disease Control
Atlanta GA 30333

January 28, 1985

Mr. Gary E. Moorehead
Staff Officer
USDA - APHIS - PPQ
Federal Building, Room 663
Hyattsville, Maryland 20782

Dear Mr. Moorehead:

Thank you for sending us a copy of the Draft Supplement to the Final Environmental Impact Statement (EIS) for the USDA Gypsy Moth Suppression and Eradication Projects. We understand that the Final EIS was approved on May 8, 1984, and our previous comments were incorporated into that document. However, to further update the Final EIS and to address subsequent comments regarding worst case analysis, the Forest Service and the USDA Animal and Plant Health Inspection Service have prepared this supplementary report. We are responding on behalf of the U.S. Public Health Service.

The risk analysis indicates that all realistic estimated doses and many worst case estimated doses of registered chemical insecticides used in gypsy moth suppression and eradication projects are below Acceptable Daily Intake (ADI) levels, and therefore are considered to be within acceptable margins of safety.

Our major concerns involve potential health hazards associated with accidental insecticide spills (aircraft and vehicular). The potential hazards associated with spills were identified within this supplement report. Fortunately, as reported, the probability of insecticide spills is "extremely low"; however, a low probability does not change the hazardous nature of the exposure. As indicated by the exposure data, and as to be expected, the dose potential is greatest for the occupationally exposed group. Among the general population, the dose potential is greater for those who do not remain indoors (estimated to be one-half of population) during spraying conditions, and for sensitive individuals who reside within the treatment areas. Cases of hypersensitivity are reportedly rare, and we recognize that it would be practically impossible to identify specific sensitive populations. We do recommend, however, that every mitigation procedure that is practical be implemented.

Such precautions should include, but not be limited to: a comprehensive worker safety program that is closely enforced; elimination of abnormal operational variations (e.g. during a 5-year period there were 3 reported cases where an area not scheduled for treatment was actually treated); implementation of a communications network that would serve to inform potentially exposed populations of spraying schedules and the procedures they should follow to protect themselves from spraying operations; monitoring operations for detecting unusual or unexpected contamination of food and water supplies; and

general procedures to be followed for potential aircraft or vehicular spills. We recognize that some of these measures were addressed within the text of the Final EIS, however, these and other relative mitigation measures, specifically with regard to worst case conditions, should explicitly be shown in a summary formation in the Draft Supplement to the Final EIS.

We appreciate the opportunity of reviewing this statement. Please send us a copy of the final document when it becomes available. If you have questions regarding our comments, please contact Mr. Ken Holt of our staff at 404-452-4161 or FTS 236-4161.

Sincerely yours,

Stephen Margolis

Stephen Margolis, Ph.D.
Chief, Environmental Affairs Group
Environmental Health Services Division
Center for Environmental Health

STATE CLEARINGHOUSE

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2/7

30 EAST BROAD STREET • 39TH FLOOR • COLUMBUS, OHIO 43215 • 614 / 466-7461

84-01-29
08 P

RCVD. FOSS 2-11-85

David E. Ketcham, Director of
Environmental Coordinator
U. S. Department of Agriculture
12th and Independence S.W., PO Box 2417
Washington, D. C. 20013

Attention: R. Max Peterson, Chief of U. S. Forest Service

RE: Review of Environmental Impact Statement/Assessment
Title: DRAFT Supplement to the Final Environmental Impact Statement of the Gypsy Mooth Suppression and Eradication Projects
SAI Number: 36-445-0011

Dear Mr. Ketcham:

The State Clearinghouse coordinated the review of the above referenced environmental impact statement/assessment.

This environmental report was reviewed by all interested State agencies. The comments received in our office have indicated there are no concerns relating to this proposal at this time.

It should be noted that your agency may still receive comments directly from a reviewing agency within the time permitted for review of this report.

Thank you for the opportunity to review this statement/assessment.

Sincerely,

Leonard E. Roberts
Deputy Director
Office of Budget & Management

LER:lw

cc: ODNR, Mike Colvin
OEPA, Barb Wooldridge

0135L

Chevron
ORTHO
Chevron Chemical Company
940 Hensley Street, Richmond, CA 94804 • Telephone (415) 231-8100
Telex: 335-459

Research
Agricultural Chemicals Division

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February 4, 1985

Gypsy Moth Suppression and Eradication Projects: Draft Supplement to the Final EIS
File No: ORTHENE EIS

Mr. Gary E. Moorehead
Staff Officer
USDA-APHIS-PPQ
Federal Building
Room 663
Hyattsville, MD 20782

Dear Mr. Moorehead:

Listed below are several comments we have on the Draft Supplement to the Final Environmental Impact Statement of the Gypsy Moth Suppression and Eradication Projects.

General Comments:

1. The "realistic dose" referred to in the document is actually the "worst case dose". This concept should be clarified in the "Introduction" section of the report so that the reader is aware of this philosophy early in the review of this document. As it now stands, this concept is not addressed until the Appendix (F-22) of this report, after the reviewer has nearly completed reading the report. a

Page 12

Paragraph 1 states "The worst case doses are 4 to 80 times below the teratogenicity NOEL for dogs (Appendix F, Table 1)".

Comment

There have been no teratogenic studies done with acephate in dogs. Likewise, there is no listing in Table 1 of a dog teratology study. b

Paragraph 1 states "Prolonged exposure to worst case doses will probably cause measurable cholinesterase inhibition and some systemic effects".

Comment

Prolonged exposure to worst case doses would not necessarily cause systemic effects since this scenario does not take into account the metabolism or excretion of acephate. c

Paragraph 1 states "This suggests that even realistic doses are overestimates". d

Comment

Realistic doses are "gross" or "extreme" overestimates. This should be so stated.

Page 13

Paragraph 1 states that "Cholinesterase inhibition is.....(treatable)".

Comment

It is not accurate to state without clarification that cholinesterase inhibition is treatable. In general, systemic effects from cholinesterase inhibitors do not manifest themselves until depression exceeds 50%. In some cases, no systemic effects have been reported in humans even when cholinesterase inhibition has reached 90%. e

The second paragraph addresses the "teratogenic NOELS of acephate".

Comment

The reference to "teratogenic NOELS of acephate" imply that there are acephate doses which are teratogenic. This is not correct as acephate has not been demonstrated to be teratogenic in animal systems. Therefore, this paragraph should be revised to correct this implication. Perhaps, the wording on Page F-6 (i.e., "In studies where no effect was observed at any dose, the highest value tested is identified as the NOEL) should be used in the teratology discussion. f

Page 14

Paragraph 2 states "Spills in water result in possible doses that exceed ADI by 10 to 18 times".

Comment

This is a "gross" overestimation we disagree with. Acephate is soluble in water and there is no reason to suspect that a chemical that is soluble in water and of an extremely low order of dermal toxicity in concentrated form (technical material) would present a hazard if spilled in the water and diluted. It is also highly unlikely that "symptoms of cholinesterase inhibiton" would result from this kind of exposure. g

Page F4

Paragraph 2 states "No IBT data were used in the development of this risk analysis".

Comment

The 2-year dog feeding study and the rat teratology on acephate referred to in Table 1 were both conducted by IBT, were both classified as valid and acceptable studies by the EPA and were both used in the development of the risk analysis. h

Page F-14

The reference to goats, rabbits and fish in the scenario discussed in the second paragraph cannot reasonably be compared to the ADI. i

Page F-16

The statement "All occupational exposure to project personnel other than observers was assumed to be equal to that received by the highest exposure group (mixer/loader)" is not supported by the data. j

Page F-27

The assumption "A reasonable expectation for fish consumption is 0.5 kg/day" is unreasonable. This corresponds to consumption of 1.1 pounds of fish per day. It is improbable any individual would eat this amount of fish (or meat) per day. k

The assumption "- no loss of insecticide from the sprayed animal as the result of excretion" is unrealistic.

Page F-69

In paragraph 1, the report states that specific individuals may be more sensitive to the chemical insecticides than the general population. As we know, this susceptibility phenomenon is addressed in estimating the ADI. l

Page F-74

Paragraph 2 as written implies that acephate produces teratogenic effects (see page 13 comments). This should be clarified. m

Page F-77

Risk of Heritable Mutations. The report states "Since there is no epidemiological data indicating a strong association....." This sentence implies that there may be an (weak) association between chemical exposure and heritable mutations. The word "strong" should be replaced by the word "any". n

I appreciate the opportunity to comment on your document.

Sincerely yours,

FXK

L. R. Stelzer, Manager
Registration and Regulatory Affairs

FXK:clv/P-7

United States Department of the Interior

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OFFICE OF THE SECRETARY
WASHINGTON, D.C. 20240

FEB 1985

ER 84/1602

RCVD. FOSS

Gary E. Moorehead, Staff Officer
USDA - APHIS -PPQ
Federal Building, Room 663
Hyattsville, Maryland 20782

Dear Mr. Moorehead:

We have reviewed the Draft Environmental Statement for Gypsy Moth Suppression and Eradication Projects and find that a number of our questions concerning the FEIS (1984) were not addressed in the draft supplement. Our concerns about the FEIS were voiced in an October 30, 1984, National Park Service letter to Mr. Thomas N. Schenarts, Area Director, Insect Disease Management Staff, Northeastern Area State and Private Forestry, U.S. Forest Service. We would like to take this opportunity to reiterate some of those comments.

1. The concept of Integrated Pest Management (IPM) as presented throughout the document is incomplete. While IPM is portrayed throughout the document as a mix of tactics stressing the "non-chemical" approaches, it can be more aptly described as a decisionmaking process consisting of monitoring, decisionmaking, and action (or treatment) components. The decision of which mix of treatment tactics to use should be based upon monitoring results that predict an unacceptable level of damage. If this level is an economic threshold, then the cost of treatment should not exceed the anticipated economic losses to resources in the absence of treatment. Aesthetic damage thresholds, while more arbitrary, may be useful in areas where economic thresholds are difficult to establish. Monitoring should include not only pre- and post-treatment effects on target populations but also effects on non-target populations, especially those most sensitive to the proposed operation. a

2. On page 11, economic losses are cited for timber and forest industries and recreational areas. How were those loss figures derived? On what assumptions are they based? Are those losses due solely to gypsy moth caused mortality? How many years of defoliation were required to produce such economic losses? What are the economic losses on a per-acre basis? Answers to these questions would be useful in a true integrated pest management approach to the gypsy moth problem. b

3. Pesticides are considered not to be harmful to the environment if acute effects are not observed. When acute effects are observed, these effects are assumed to be of a temporary nature. The statement on page 59 (lines 3-5), ". . . populations of the nonsensitive forms adjust the overall community numbers to counteract the effects . . . ." is puzzling from an ecological standpoint. Is the assumption made that the number of organisms, regardless of type, is the most important property of the community? Since sub-acute and chronic effects of pesticides on population, community, or ecosystem level properties and functions (e.g., diversity, age structure, nutrient cycle, energy flow) have not been adequately tested, it is premature to assume chemical insecticide treatment will not result in any irreversible or irretrievable adverse environmental impacts. c

4. No discussion is presented on the rate or pattern of spread by gypsy moth populations. Such information would be useful in making predictions about the dispersal of the population from a given area and would be valuable in an overall IPM approach. Are there scientifically valid studies available to support these types of predictions or are current predictions solely speculative in nature? What effect does host plant quality have in the dynamics of the population? Can it influence the rate and pattern of population dispersal or qualitative characteristics of the gypsy moth population? d

5. The Affected Environment section of the FEIS (page 28) does not adequately discuss the various management objectives for land areas. For example, areas could be managed for: timber production, recreation use, forested communities or as natural areas undisturbed by human activities. Are gypsy moth defoliation and gypsy moth caused tree mortality different for areas with different management objectives? This information would be useful to land managing agencies in selecting strategies for gypsy moth control. e

6. The draft supplement should include at least general guidance on the direction that site-specific plans should take in the proposed monitoring and control/non-control actions involved. The programmatic EIS should be edited/rewritten to clearly state the inevitability of spread of the gypsy moth; to depict through maps, acreages, and narrative, the history and mechanics of the "leading edge" of the infestation; to include the criteria used in establishing "regulated" or "unregulated" areas in relation to the "leading edge"; and most importantly, to clearly state goals and the differences between monitoring and control goals within and outside of the areas where gypsy moths have become permanently established. f

7. Annually, the U.S. Forest Service treats only 10% of the area infested by gypsy moth (page 14 of FEIS). Criteria used by the Forest Service for selecting such a proportionately small treatment area should be included and fully discussed in the supplement. g

We hope these comments are helpful to you.

Sincerely,

Bruce Blanchard, Director
Environmental Project Review

UNITED STATES ENVIRONMENTAL PROTECTION AGENCY
WASHINGTON, D.C. 20460

FEB 15 1985

Mr. Robert L. Williamson
Director, National Program Planning Staff
USDA Animal and Plant Health Inspection
Service, Federal Building - Room 648
Hyattsville, Maryland 20782

Dear Mr. Williamson:

In accordance with our responsibilities under the National Environmental Policy Act (NEPA) and Section 309 of the Clean Air Act, the Environmental Protection Agency (EPA) has reviewed the Draft Supplement to the Final Evironmental Impact Statement (EIS) for Gypsy Moth Suppression and Eradication Projects.

The draft supplement incorporates additional information and analyses in Appendix F concerning potential human health risks associated with the use of the insecticides: acephate, carbaryl, diflubenzuron and trichlorfon in gypsy moth suppression and eradication projects. Revisions have also been made in the Chemical Insecticides Section of the Environmental Consequences Chapter in order to reflect the revised risk analyses. We note that the project alternatives presented in the final EIS remain unchanged, with the preferred alternative identified as the Integrated Pest Management (IPM) alternative.

We believe that the human exposure estimates and scenarios presented in Appendix F are based on reasonable assumptions and result in prudent estimates of the risks associated with gypsy moth suppression and eradication activities. We have, however, enclosed technical comments and corrections of general statements and specific figures concerning potential health effects which will help to clarify certain elements of the risk analyses. Specifically, these changes would lower the margin of safety and no observable effect level (NOEL) for diflubenzuron which may result in changing the overall risk estimate for this pesticide. In our judgment, however, these changes do not alter the conclusion expressed in our letters of February 23, 1984, May 7, 1984 and July 30, 1984. Based on the information currently available and the identification of Integrated Pest Management as the preferred alternative, EPA continues to have no objections to the Gypsy Moth Program as proposed, provided that all appropriate mitigation measures are implemented. All activities would of course have to be conducted in compliance with applicable Federal and State law. Accordingly, EPA has rated this Draft Supplement "LO" (Lack of Objections). A copy of our rating system is enclosed.

Rec'd from Messenger EPA
9 a.m. 2/19

I hope that these comments will be useful to the preparation of the final supplement to the EIS. Please do not hesitate to contact David Durham (475-8789) of my staff if we may be of further assistance.

Sincerely,

Allan Hirsch
Director
Office of Federal Activities

Enclosure

DETAILED COMMENTS ON THE DRAFT SUPPLEMENT TO THE GYPSY MOTH FINAL ENVIRONMENTAL IMPACT STATEMENT

Carbaryl

1. Page 17 states that EPA "has requested the registrants to repeat the teratology (sic) in the beagle dog."

   The Agency issued a Registration Standard for carbaryl in April 1984, which included an additional teratology study in dogs as a condition of continued registration of products containing carbaryl. However, a registrant of carbaryl requested that EPA reconsider this requirement, and stated that such a study was unnecessary. The Agency is considering this request in relation to the data available on teratogenic potential of carbaryl, and a decision is expected on the need for the requested additional studies within several weeks. a

2. There is no explanation given for the origin of two quantitative factors used in the analysis of carbaryl risks. On page F-60, the relationship of *in vivo* to *in vitro* yield of n-nitrosocarbaryl is not explained. There is also no source given for the derivation of the cancer potency slope (the value of B in the risk estimate). We suggest that these sources be cited. b

Trichlorfon

1. Page F-9 bottom paragraph, correctly states there is uncertainty about the potential carcinogenicity of this chemical. To clarify this, the last sentence on the page should be expanded to add "---, but the available data are inadequate for a quantitative risk assessment for oncogenicity, and thus further studies are needed." You may be interested to know that the Agency is currently developing a Registration Standard for this chemical, which will probably require additional studies to address the oncogenicity issue and to reevaluate the ADI and associated tolerances for this pesticide. The standard is expected to be issued this spring. c

   The studies cited on page F-10 (top paragraph) are judged to be inadequate by EPA for the purposes of EPA's Registration Standards. They may, however, help to emphasize the uncertainty present regarding the oncogenicity potential of trichlorfon. Therefore, the assumption on page F-10 that trichlorfon is a carcinogen represents a prudent approach to analyzing the potential risks associated with trichlorfon's use. d

2. As you note on page F-13, EPA's Carcinogen Assessment Group (CAG) does not use the simple linear model cited on F-10 for evaluating trichlorfon or nitrosocarbaryl, because they believe that sampling error associated with B is high. CAG utilizes a 95% bound on the linear coefficient (Q1)in a multistage model. However, we do agree with the statement that risk assessments based on simple linear models, such as those used in this document, generally provide conservative estimates of the risks (that is, they estimate the upper limit for the risk present). e

3. Page F-53 (top paragraph) refers to a NOEL (1.25 mg/kg/day) for trichlorfon listed in Table 4 (p.F-95). Please note that the study cited to support this NOEL is judged to be inadequate for EPA's Registration Standard purposes. In our evaluation system, this study is rated as supplementary, meaning that it was not conducted according to current standards, although it is not necessarily invalid, and its information is considered to be useful. The text should note this qualification, and that it is being used as the best indication of the NOEL currently available. f

Acephate

1. There is some indication of potential oncogenic activity for acephate, which the Agency is currently in the process of evaluating. Thus, it may be advisable to note that this possibility is under evaluation at the present time, rather than simply listing the cancer potency for acephate in Table 7 as "none." g

   The Agency is developing a Registration Standard for acephate, due to be issued this summer. The evaluation of possible oncogenic effects evidence for acephate will be completed prior to completion of the standard, so that a decision can be made on an appropriate regulatory position regarding pesticide products containing acephate.

2. In Table 1, the first study listed, giving a NOEL of 5ppm for cholinesterase (ChE) inhibition in the rat, is an invalid IBT study considered to be inadequate for establishing a NOEL, and should not be cited. There is another study (the fourth) in Table 1, however, which established a NOEL of 5ppm for ChE inhibition in the rat; it would be more appropriate to cite this study only. h

Diflubenzuron

1. In several places, the figure cited as a NOEL for diflubenzuron is not accurate (p.20, p.F-53 and Table 3 the 80 week mouse study). The correct characterization is that the agency determined a NOEL of 1.1 mg/kg/day by regression analysis of 13 week and 80 week mouse feeding studies. The ADI for diflubenzuron thus reflects a 100-fold safety factor, and not a 200-fold factor as currently stated. The top paragraph of p.F-53, and the references to the safety factor on F-69 and F-74 should be corrected, as well as Table 3. i

   Correspondingly, Table 10, the relationship of expected realistic and worst-case doses to the ADI and NOEL for diflubenzuron, will have to be recalculated. However, it appears that significant margins will still exist between the dose levels and the ADI and NOEL.

Miscellaneous

1. On pages F-52 and F-69, acceptable daily intake (ADI) is described as an "internationally established standard" and an "international exposure standard". These phrases imply a formal status which is not true. ADI can be described as an internationally recognized concept used in evaluating exposure to pesticides. j

2. On page F-70 and again on page F-71 there is reference to "daily consumption of contaminated meat, vegetables ...". If this is intended to refer to residues which occur in foods as a result of pesticide use, that should be clearly stated and not described as "contamination".

   Pesticide residues in foods are regulated by the establishment of tolerances, which are legally acceptable maximum residue levels. Residues above tolerance levels or for which no tolerance is established render a commodity adulterated under the Federal Food, Drug and Cosmetic Act. The Food and Drug Administration (FDA) enforces tolerance requirements for most agricultural commodities, except for meat, poultry and some eggs, which are sampled by the U.S. Department of Agriculture. k

PESTICIDE HAZARDS CLEARING HOUSE
BOX 723
NORTH CAPE MAY, NEW JERSEY 08204

2/14/'85

Gary Moorehead, SO
USDA-APHIS-PPQ
Hyattsville, MD

Dear Sir,

Upon reading over our comments on the g. moth Draft Supplement To The Final Environmental Impact Statement I noticed that our Secretary made some mistakes on the title page & Preface.

If possible, we would appreciate it if you could substitute these corrected pages for the pages in the comments.

Sincerely,

Kenneth Hobbs-Director

KH/sc

PESTICIDE HAZARDS CLEARING HOUSE
BOX 723
NORTH CAPE MAY, NEW JERSEY 08204
February 1, 1985

COMMENTS OF THE PESTICIDE HAZARDS CLEARINGHOUSE ON THE 1985 U.S. DEPARTMENT OF AGRICULTURE GYPSY MOTH SUPRESSION AND ERADICATION PROJECTS DRAFT SUPPLEMENT TO THE FINAL ENVIRONMENTAL IMPACT STATEMENT

Kenneth Hobbs, Director

Preface

These comments on the 1985 U.S. Department Of Agriculture Gypsy Moth Supression And Eradication Projects Draft Supplement To The Final Environmental Impact Statement (abbreviated to "DS" below) are preliminary in nature. More extensive comments with full documentation of references are planned in the comments on the Final Supplement To The Final Environmental Impact Statement.

The Pesticide Hazards Clearinghouse is a non-profit organization which acts as a clearinghouse for information on hazards of pesticides. Valid information from all sources is welcomed and will be added to the reference files. Correspondence to the organization's box will be answered within about 6 weeks. The Pesticide Hazards Clearing House operates from April through October.

PESTICIDE HAZARDS CLEARING HOUSE
BOX 723
CAPE MAY, NEW JERSEY 08204

2/15/'85

Gary Moorehead
USDA,APHIS,PPQ
Hyattsville MD

Dear Sir,

We apologize for this, but we have found that our stationary used an erroneous adress for our organization box. Our previous submissions of material to you bore the erroneous adress "North Cape May". The actual adress is "Cape May".

We have retyped another title page to our Comments On The USDA Gypsy Moth Supression & Regulatory Projects Draft Supplement To The Final Environmental Impact Statement, using stationary with the correct adress.

Sorry For The Bother,

Kenneth Hobbs-Director

KH/sc

PESTICIDE HAZARDS CLEARING HOUSE
BOX 723
CAPE MAY, NEW JERSEY 08204
February 1, 1985

COMMENTS OF THE PESTICIDE HAZARDS CLEARING HOUSE ON THE 1985 U.S. DEPARTMENT OF AGRICULTURE GYPSY MOTH SUPRESSION AND ERADICATION PROJECTS DRAFT SUPPLEMENT TO THE FINAL ENVIRONMENTAL IMPACT STATEMENT

Kenneth Hobbs, Director

TENNESSEE VALLEY AUTHORITY
KNOXVILLE, TENNESSEE 37902

32

FEB 11 1985

RCVD. FOSS 2-19-85

Mr. Gary E. Moorehead, Staff Officer
U.S. Department of Agriculture
APHIS - PPQ
Federal Building, Room 663
Hyattsville, Maryland 20782

Dear Mr. Moorehead:

The Tennessee Valley Authority has reviewed the Draft Supplement to the Final Environmental Impact Statement (FEIS) on Gypsy Moth Suppression and Eradication Projects. We believe that the U.S. Forest Service and the Animal and Plant Health Inspection Service have adequately addressed those additional comments raised subsequent to the issuance of the FEIS. Thank you for the opportunity to review the document.

Sincerely,

Martin E. Rivers, Director
Environmental Quality

COMMENT LETTER INDEX

Number	Comment Letter
1	Maryland Department of State Planning
2	North Carolina State Clearing House
3	Oregon State Clearing House
4	Ohio State Clearing House
5	Tennessee Historical Commission
6	Iowa State Clearing House
7	Rhode Island Office of State Planning
8	Missouri State Clearing House
9	Washington Department of Ecology
10	Oregon State Clearing House
11	National Network to Prevent Birthdefects
12	Northwest Coalition for Alternatives to Pesticides
13	Pesticides Hazards Clearing House
14	Oregonians for Food and Shelter
15	North Carolina State Clearing House
16	South Carolina Office of the Governor
17	National Coalition Against the Misuse of Pesticides
18	New Jersey Department of the Public Advocate
19	Virginia Council on the Environment
20	Kenneth and Janet Nolley
21	Glen and Elaine Olsen

The remaining comment letters, 22-32, were received after the close of business February 4, 1985, the final day of the 45-day comment period.

22	New Jersey Coalition for Alternatives to Pesticides
23	Pennsylvania Intergovernmental Council
24	The Resources Agency of California
25	United States Department of Health and Human Services
26	Ohio State Clearing House
27	Chevron Chemical Company
28	United States Department of Interior
29	United States Environmental Protection Agency
30	Pesticides Hazards Clearing House
31	Pesticides Hazards Clearing House
32	Tennessee Valley Authority

Comment Letters and Responses to the
Draft Supplement to the FEIS

Introduction

Comment letters on the draft supplement to the FEIS were received from 32 individuals, agencies or organizations. Ten comment letters were received after the close of business on February 4, 1985, the final day of the 45 day comment period. The Forest Service and APHIS nevertheless attempted to address all of the comment letters received, whether received within the comment period or not. The agencies were not required to do this by NEPA. All of the comment letters received, including the late ones, are part of the administrative record, and will be considered by the Responsible Officials of Forest Service and APHIS in the final decision-making process.

All of the comment letters are numbered on the upper right corner. Each substantive comment that was considered and addressed is identified by an alphabetical letter located on the right side of the comment letter.

Responses to comment letters received are as follows:

Letter No.	Comments	Response
1		No response required.
2		No response required.
3		No response required.
4		No response required.
5		No response required.
6		No response required.
7		No response required.
8		No response required.
9		No response required.
9-A		Corrections made.

Letter No.	Comments	Response
10	a	These reports represent observations by astute clinicians reported in scientific journals. We feel adequate precautionary statements were included in the *B. t.* discussion regarding the fact that these were not studies.
	b	A toxicological discussion of *B. t.* is covered on pages 69-72 of the FEIS. This discussion covers exposure rates at which no effects have been observed. Since no acute or chronic toxicity was observed in any of the tests, no true NOELs can be established. EPA did not furnish us with an ADI level for *B. t.*
	c	Comment noted. These terms are defined in the glossary.
11	a	The Department on August 23, 1984 published in the Federal Register (49 FR 33471-33472) a notice of its intent to supplement the 1984 FEIS. The notice stated that no further action would be taken under that FEIS until supplemented. The Department's notice of availability of the Draft Supplement to the FEIS appeared in the Federal Register (49 FR 49649-49650) on December 21, 1984. In neither of these notices was any reference made to "discarding" the 1984 FEIS. Copies of this FEIS have been available upon request since its publication.
	b	In recognition of the difficulty in presenting this very technical topic, a writer editor was added to the writing team for the express purpose of improving the readability of the document. For positive comments on this aspect of the document, see letters 14, 19B, 19C, and 29.

Letter No.	Comments	Response
	c	See response 11a.
	d	Potential adverse health effects of carbaryl and corresponding no effect levels are summarized in Appendix F, Table 2. Additional data requested by EPA from the manufacturer are discussed on F-8; also see EPA comment letter number 29a. We know of no studies that provide data linking Dimilin to leukemia. However, increased methemoglobin and sulfhemoglobinemia effects have been reported to EPA and mentioned by other commentors. All effects are dose related. A discussion of this effect has been added to the text beginning on page F-10. The Department feels the potential hazards, exposure, and health risks associated with the use of acephate, carbaryl, B. t., diflubenzuron, and trichlorfon are thoroughly discussed in this FEIS.
	e	Your opinion noted. Your statements relating to the suppression and eradication projects are not based on fact. Projects which historically have treated less than 10 percent of the defoliated area and which now rely heavily on B. t. and diflubenzuron (both which have little if any impact on parasites or predators) are not "designed" to kill natural predators and promote faster spread. Additionally, the gypsy moth budgets in both the Forest Service and APHIS have not been increasing, but have been stable or declining.
	f	See the FEIS for a discussion of biological controls and the alternatives (pages 14-24 and 32-76).

Letter No.	Comments	Response
	g	See response 11a. The draft supplement proposed changes in the body of the 1984 FEIS, which necessitated merging these changes into the document before one could reasonably comment on these proposed changes. Therefore, comments on the 1984 FEIS, as impacted by these proposed changes, were not precluded.
	h	The Department has provided the appropriate public comment and review period on this document as required by NEPA.
	i	Each route of exposure was discussed separately (in the Analysis of Exposure section beginning on p. F-25) primarily to ease calculations of dose and the understanding of the dose estimates. These separate doses were added to evaluate risk of the composite dose.
	j	The Department has made no claim of reliance on EPA registration as proof of the safety of any insecticide discussed. The rationale and basis for the risk analysis are discussed on pages F-2 through F-5. All scientific data on possible adverse health impacts that we were able to find in an extensive literature search (see F-7 for description), or that were made available to us by EPA or the registrants are listed in Tables 1 through 4 of Appendix F.
	k	The Department disagrees with your opinion and feels that the FEIS meets the requirements of NEPA. Also see previous response 11h on this subject in your letter.
	l	Comment noted. See response 11h.

Letter No.	Comments	Response
12	a	The risk analysis does not deny the hazard or risk associated with the use of pesticides. Both risk and the probability of occurence are discussed on pages F-70 to F-104. In our opinion, this meets the requirements of NEPA and provides the decisionmaker with the proper tools for making a reasoned decision. Accidents result in the greatest level of insecticide exposure and therefore present the greatest risk of adverse health impacts to exposed workers or the general public. The analysis states that since this type of exposure is very close to the dermal LD_{50} for acephate, carbaryl, or trichlorfon, symptoms of acute toxicity would occur. These symptoms could range from dizziness, headache, weakness, muscle twitching, nausea, vomiting and diarrhea for lower levels of exposure to sudden unconsciousness, toxic psychosis, or respiratory depression which may be fatal.
	b	See response to 11d for discussion of methemoglobenemia and sulfhemoglobinemia. The concern about 4-chloroaniline was investigated by EPA when further oncogenicity studies were requested. The new tests satisfied this concern. Metabolites of acephate, potentiation, and bioconcentration are discussed on pages F-38, F-101, and F-39 to F-40 respectively.
	c	This argument objects to exposure or insecticide residue information for carbaryl, diflubenzuron, and trichlorfon being extrapolated to cover initial acephate residues or

Letter No.	Comments	Response
		exposures. As noted on F-27 carbaryl has the most complete data base. However, the assumption that one pound of any insecticide, applied in similar methods, will result in one pound of insecticide being deposited on an acre of ground independent of the insecticide seems logical. The chemistry of the individual insecticide would then determine dermal absorption rates, bioaccumulation, or environmental fate used in the analysis. At the time the Draft Supplement was written, some information on acephate's persistence in water, plants and animal tissues was overlooked. This information has been added to the analysis, see pages F-41 to 42. This data was consistent with the values for acephate (except for the dietary component) that were determined by extrapolating data for carbaryl and trichlorfon.
	d	We followed the procedures for mutagenicity risk assessment published by EPA (1984 FR 49:227). In this procedure, the overall weight of evidence is considered in determining mutagenic risk. Just because a chemical is mutagenic in Salmonella (bacteria), it does not mean it will be mutagenic in higher animals. The error concerning unscheduled DNA synthesis in cultured human fibroblasts has been noted and corrections were made.
	e	The Seiler (1977) citation was an error that has been corrected. The proper references and documents were supplied by the EPA. See page F-14 for the correction. Because of your concern and information provided in the EPA comment letter (see 29) a cancer risk analysis for acephate has been added to Appendix F.

Letter No.	Comments	Response
	f	As noted on pages F-7 and F-8, the analysis of risk associated with using acephate, carbaryl, diflubenzuron or trichlorfon is independent of the EPA's registration process. Use of IBT data were discussed on F-7. No IBT data that had been judged to be invalid by EPA was used in the risk analysis. Data on acephate residue on broccoli and lettuce has been added (see response 12n). It is important to note that consumption of food or water are on a per day basis in order to calculate dose to humans in terms of mg/kg/day. This allows a comparison of maximum human doses to NOEL values determined from animals. For the cancer analyses, where people could consume food or water containing insecticide residues for more than one day, the persistence of the specific insecticide dictated both the number of days that food would be consumed and the residue level.
	g	The basis for the 10 percent absorption rate is discussed on F-29. The concept for estimating dermal absorption was in the book that was edited by Eto (1977). Acephate data was one of the data sets used which gave a value of 8.4 percent. This value was scaled up to 10 percent.
	h	If acephate were applied at the same application rate, there is no reason not to expect that acephate would be deposited at the same rate per hour as carbaryl. This value is no longer used for the worst case exposure (see F-32). However, the study is still used to support the values used in the analysis.

Letter No.	Comments	Response
		The reason the 0.029 mg/kg/day values was mentioned is to alert the decisionmaker and public that if a resident acted like an observer, the worst case value for the observer would be applicable. The analysis includes a discussion of an observer that receives a direct insecticide application and then consumes food containing insecticide residues. It is important to note that the dietary component more than doubles the exposure in the worst case.
	i	The basis for using 10 percent dermal absorption for rabbits is given on F-33. This discussion also refers back to F-29 because the value used for humans came from studies using test animals. It must be noted that this value is only used to calculate the worst case residue level of acepate that might be in rabbit meat. Actual test data show that true residues are below the detection limit. Therefore, the worst case estimate of 0.25 ppm (0.34 x .75) is at least 5 times higher than any value that has been measured.
	j	The range of 1 to 100 ppm for insecticide residues is for residues on the type of plants that animals might eat (see F-35). Since humans eat different plants or plant parts, studies dealing with leafy vegetables were reviewed to determine the range of residues that humans could possibly eat (see F-42 to F-43).
	k	This data has been added to the analysis, pages F-42 to F-43. The Chevron (1973) report referred to in the comment states that acephate is "readily degraded by plants. The half-lives observed are generally 5-10 days ..." In our opinion this is short lived.

Letter No.	Comments	Response
	1	As stated on F-40, 10 percent assimilation is assumed. We were not able to find any data to support this value or the assumption that there was no insecticide loss due to excretion. It must be remembered that these values were used only for worst case estimates. Since these estimated worst case values exceed residue values that have been determined from actual feeding studies by 5 to 1,000-fold, the accumulated assumptions obviously overestimate risks.
	m	We were not able to obtain the Tucker and Stevens (1978) reference from EPA before this document was finalized. EPA has been contacted and the reference in question will be made part of the administrative record once it is received by the Department. However, data reported by LOTEL (1975) pertaining to water residues were included on F-42. This study reported a maximum of 0.10 ppm acephate in water one day after treating an area for gypsy moth control. Residues fell below detection limits within 5 days. In addition, the water samples, as described in your comments, were removed from the pond and stream and held at 9°C. This procedure would prohibit natural degradation through normal environmental processes.
	n	Data for acephate residues on lettuce and broccoli from the Chevron (1973) study have been added to the analysis on page F-43. It is important to note that the residue levels resulting from a 2 lb/acre application (2.5 times the registered gypsy moth rate) of acephate ranged from 4.3 to 12.4 ppm 3 days after treatment. These values are well below the range of 10 to 50 ppm used in the analysis.

Letter No.	Comments	Response
		However, since these residues cannot be washed off the leaf surfaces once the acephate is absorbed on or into the plant, the value of 11.4 ppm was used after washing. It is important to note that persistence of the residues noted on responses 12m and 12n are only used when calculating cancer risk. When realistic or worst case doses are compared to ADIs or NOELs, the maximum dose (without any degradation) is used in the comparison. Therefore, persistence is irrelevent in this situation. In the cancer calculation, persistence of residues on meat, water, or vegetables were lumped together to simplify calculations. In that case, it was assumed that all residues, for a specific insecticide, would degrade at the rate of the most persistent residue. These time periods were 20 days for acephate, 14 days for carbaryl, and 60 days for trichlorfon.
	o	In trying to determine the worst case variables, we tried to be realistic. The fact that the 2x mixing error did occur lends credibility to the risk analysis. However, it is important to note that the 2x Dimilin dose of 4 oz that resulted from the mixing error is equal to the realistic dose that was used in this analysis (0.06 lb ai/acre). In other words, the impacts or exposures were only those that would result from the realistic dose so the worst case did not actually occur.
	p	As pointed out on pages F-66 and F-87, the concept of comparing human exposure levels to ADIs or NOELs to establish acceptable levels of exposure has been published in many toxicology textbooks. In addition, EPA

Letter No.	Comments	Response
		recently published guidelines for assessing development risks using the same concept (49 FR 46324 to 46331). General health effects that can be measured with no effect threshold levels are discussed beginning on pages F-9 and F-67. NOELs for specific effects are also listed in Tables 1 through 4 in Appendix F. Specific responses that were measured in the experiments have been added to Tables 1 to 4 in order to provide a clearer understanding of the type of threshold responses the different insecticides may cause. It is important to note, however, that the two cases where B. t. caused diseases in humans were discussed because B. t.'s mode of action is to cause bacterial diseases in insects. Acephate, carbaryl, and trichlorfon are central nervous system toxins with cholinesterase inhibition being the principle mode of action. Therefore, it was important to discuss cholinesterase inhibition for these insecticides.
	q	As noted in Table 1, F-7 and F-8, the NOELs for acephate are not based on incomplete data.
	r	The reason for presenting the safety factors that were used by EPA in establishing the ADIs for acephate, carbaryl, diflubenzuron and trichlorfon was to provide the decisionmaker and other readers with a yard stick to be used when comparing NOELs to estimated human exposures. The National Academy of Science (NAS 1977) provides one of the best discussions of safety factors. Safety factors can be 10, 100, or 1000 depending upon the adequacy of the data base. The acephate ADI incorporates a safety factor of 10 because cholinesterase

Letter No.	Comments	Response
		depression, the only health impact that was observed in the multi-year study, is a well documented effect with ample data on human response. All ADIs were provided by EPA with the exception of trichlorfon. The Registration Standard for trichlorfon concluded that the study used to establish the ADI (0.125 mg/kg/day) was inadequate by today's standards. Therefore, the lower ADI established by WHO was used in the risk analysis. Since EPA had not expressed a similar concern about the carbaryl ADI (see USEPA 1984a and 1984g), it, and not the WHO value, was used in the analysis.
	s	Only worst case doses that include a dietary component exceed the ADI. This is discussed on pages F-68 and F-87. The next highest NOEL for cholinesterase depression (the dog data) was discussed to show readers the variation in NOEL for the same health effect measured in different species. If humans respond more like dogs than rats, then the worst case exposure would be lower than the NOEL by a margin equal to the safety factor.
	t	Most human (excluding sensitive individuals) can tolerate cholinesterase depressions of 50 percent before adverse symptoms are observed. This response is reversible once exposure has been eliminated. Since this was the lowest NOEL for acephate, it was important to compare the worst case doses to NOELs for other possible adverse health effects.
	u	As stated earlier in response 12r, the discussion of safety factors was included in the risk analysis

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		in order to put the comparison between human exposure and NOEL into perspective. It is only suggested that the safety factors used by EPA in establishing ADIs provides a margin of safety that might be acceptable to the decisionmaker. The final decision about acceptable margins of safety or risk levels are made by the Responsible Officials of the Forest Service and APHIS. For the Department's comment regarding the carcinogenicity uncertainty of 4-chloroaniline, see response 13kk.
	v	Since the dermal exposures resulting from accidents approach but do not exceed the dermal LD_{50} values for acephate, carbaryl and trichlorfon, adverse health impacts most certainly would occur (as stated F-91). However, these impacts can not be quantified because the slope of the LD_{50} line is not available in the scientific literature. In order to address this data deficiency, the symptoms of cholinesterase inhibition have been added to the discussion on page F-91.
	w	The ADIs were left unaltered because the values were set by either EPA or WHO. We felt that the reduction of the NOELs by 100, coupled with margins of safety that may be acceptable to the Responsible Official (e.g. 100) provide a margin of safety for sensitive populations of 10,000. The review of scientific literature provided to us by Dr. Edward Calabrese showed that while the range in human variation in the metabolism of various xenobiotics may approach or even at times exceed a factor of 1000x, the vast majority of responses reviewed

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		seemed to clearly fall within a factor of 10. Thus, the commonly used safety factor of 10, which is designated to take into account human variability in response to toxic agents appears to provide protection for up to about 80 to 95 percent of the public depending upon the specific situation. Dr. Calabrese did not find any studies that were specific to carbaryl. However, Dr. Calabrese provided us with a draft publication dealing with carbaryl-induced methemoglobin formation and glutathione depletion in sheep with G-6-PD deficient red blood cells. The language beginning on page F-99 has been rewritten to more clearly express the risk to sensitive populations.
	x	The information reported by Statham and Lech (1975a, 1975b, and 1976) has been added to the discussions dealing with synergism. However, these studies reinforce the use of the 10x safety factor based on the interaction of smoking and exposure to asbestos. Since this value is higher than any documented effect, we believe the discussion on synergism is adequate. The reference to EPAs tank mix notice was included to show that no pesticide potentiation nor synergistic effects were found by EPA after examining considerable data over a number of years. If potentiation did not occur at the high pesticide concentrations that exist in the mix tank, it is very unlikely that synergism or potentiation would occur at the extremely low environmental concentrations that result from the use of the insecticides in gypsy moth control projects.

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	y	Since there is no evidence that either carbaryl, acephate, diflubenzuron or trichlorfon accumulate in the environment, the example of DDT is completely inappropriate.
	z	The first application would be somewhat degraded by the time of the second application, 14 days later. The resulting exposure dose would be somewhere between calculated realistic dose (1.1x) but would not exceed a worst case dose (i.e. 2x) unless a mistake in mixing or application were to occur. The decisionmakers can determine the affects in Table 10 by simply doubling the realistic dose values.
	aa	Data from the study reported by Lijinsky and Taylor (1976, Cancer Letters, 1:275-297) was not used in calculating the cancer potency of N-nitrosocarbaryl because the study did not include an untreated control. Without a control, there was no way to determine the level of spontaneous tumors that exist in the test animals. Furthermore, the 29 percent cancer incidence reported by Eisenbrand et al. (1976) includes autopsy reports of the presence or absence of lesions in the animals that died before the study's end. The cancer risk analysis is based upon a 70-year lifetime; consequently, the model accounts for the longer life span of children following exposure.
	bb	The yields of N-nitrosocarbaryl are based on both *in vivo* and *in vitro* data. Since this was the only data available, the use of other yield values would be pure speculation, contrary to existing scientific data.

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	cc	The discussion on the risk of heritable mutations has been changed, see pages F-97 to F-98. The statement about the inability to measure heritable responses has been removed. An estimated worst case incidence of mutation has been added which was based on the incidence of cancer. The cancer rate was used because the incidence of mutations follows basically the same mechanism as cancer for trichlorfon. Other options to mitigate risk have also been included in the FEIS.
	dd	To help clarify the risk analysis, a flow chart depicting the risk analysis process has been added to Appendix F. Further discussion of data gaps and scientific uncertainties have also been added to the FEIS. In addition, a matrix depicting the relationship of the doses in the risk analysis to human health effects has been added to the FEIS.
13	a	Correction made.
	b	Correction made.
	c	Corrections made.
	d	Correction made.
	e	Data from the full study have been added to F-14.
	f	We agree in essence, but we had to look at the data that was available to us. We tried to differentiate between data from U.S.- or foreign-produced insecticides.

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	g	The fact that the cancer incidence in the untreated animals was twice as high as that in the treated animals raises the question of whether trichlorfon should even be considered a suspect carcinogen. The use of this data and the assumption that the cancer incidence in the untreated animals is zero does not invalidate the analysis, rather it constitutes a worst case analysis where uncertainty exists.
	h	Carbaryl is metabolized in the body to form conjugates of 1-napthol which are then excreted. The cited studies measured the 1-napthol levels in the urine.
	i	The Department recognizes that the carbaryl exposure data used for the mixer/loader group includes exposure to pilots from sources other than gypsy moth projects. The data was therefore considered to represent an upper limit of the realistic exposure that the mixer/loader group is likely to receive. The data is not invalid, but rather extremely conservative (an overestimate).
	j	Gypsy moth projects may very well be conducted in the evening as well as the morning hours. The data is therefore considered to represent an upper limit of the realistic exposure that project workers are likely to receive. The data represents an overestimate.
	k	The error has been corrected.
	l	Correction made.

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	m	Correction made.
	n	Upon reanalysis of Rickard and Dorough (1979), it was found that the value 0.002 referred to *in vivo* yield and not the relationship of *in vivo* to *in vitro*. Therefore, the *in vitro* yield is not necessary. Appropriate changes have been made in the text.
	o	Correction made.
	p	Correction made.
	q	Correction made.
	r	Correction made.
	s	Correction made.
	t	Correction and addition made.
	u	Correction and addition made.
	v	Deletion and addition made.
	w	Misspelled word corrected. The two doses represent two different methods of exposure (gavage vs. dietary inclusion). This has been clarified in Table 2.
	x	Misspelled words corrected. Maternal weight loss is not a teratogenic effect. The NOEL identified is for omphalocele in that there was no significant difference in the occurrence of this effect between test and control animals. While reviewing Murray et al. it was noticed that rabbits were fed by gavage only. Therefore, the 5660 ppm dietary dose has been removed.
	y	Addition made.

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	z	There is no reference to mutagenicity in this study, but it does deal with reproductive effects and is so noted in Table 2. A LOAEL of 10 mg/kg/day was added for reproductive effects (dilated uterine glands) in third generation pups.
	aa	Addition made.
	bb	Defects of the cervical vertebrae were a significant terata. The missing organs (kidney and genitals) were only seen in two fetuses. There was no discussion as to whether this was a significant finding.
	cc	The NOEL value for dogs was provided by EPA. The neonatal functional deficits and excessive crying were not signficant in Smalley et al (1968).
	dd	A LOAEL of 1000 mg/kg/day was added since a dominant lethal effect was detected.
	ee	Comment noted and footnoted in Table 4.
	ff	For the mouse, the 400 represents low fetal weight; not a true terata, but an adverse reproductive effect. The 500 mg dose produces terata in the form of cleft palate, and reproductive effects in terms of low fetal weight. It is so noted in Table 4.
	gg	For the rat the change was made. For the hamster the 200 is a true NOEL. At 300 mg/kg/day there is reduced maternal weight gain and reduced fetal weight. At 400 mg/kg/day various malformations were noted and are true terata.
	hh	Comment noted, no response necessary.

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	ii	This is a complex situation but true in the general sense. However, as noted by EPA (1984k) no models exist for extrapolating risk for developmental toxicology. Therefore such risks are evaluated using thresholds, safety factors, and margins of safety as was done in Appendix F.
	jj	This comment provides an additional discussion about how to evaluate various *in vitro* mutagenicity tests to predict whether a chemical may be a human mutagen. To further clarify this point, Table 18 has been added to Appendix F. This table lists the various *in vitro* tests and ranks them as to their ability to predict cancer or mutations.
	kk	Discussions concerning the mutagenic and carcinogenic potential of 4-chloroaniline have been added to Appendix F, see F-12 and F-21. Since there was uncertainty about whether 4-chloroaniline is carcinogenic, cancer risks were estimated assuming that it was a carcinogen. The only reference found by USDA concerning mutagenicity of 4-chloroaniline was a report of positive mutagenicity in Ames tests in the diflubenzuron Decision Document (USEPA 1979 in Appendix F).
	ll	In reviewing whether carbaryl might pose a mutagenic risk, the Department relied on the Carbaryl Decision Document (USEPA 1980a). The conclusion of this review was that "carbaryl is not intrinsically a potent mutagen in the reported studies, and probably acts as a weak mutagen only."
	mm	Comment noted, no response necessary.

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	nn	Comment noted, no response necessary.
	oo	If the Knaack et al. (1968) study referred to in this comment is the study titled "The metabolism of carbaryl in man, monkey, pig and sheep" in J. Agr. Food Chem. 16: 465-470, then the Department reviewed the same publication. This study is a metabolism study that dealt more with the nature of the urinary metabolites of carbaryl than in finding how fast carbaryl or its metabolites are excreted from the body. The study does not report excretion rate other than to show a single graph. The 24 hour cumulative percent was a little greater than 25 percent. However, the authors reported that recovery by their analytical method was less than that obtained with colorimetric techniques (25 percent vs. 37.8 percent). Since recovery rate is obviously dependent upon the analytical method, it seemed appropriate to use the multiplier of 3 suggested in the (SCESC 1978) report since these were the people doing the chemical analysis.
	pp	The only reference by Mittleman that the Department was able to locate was an internal EPA memo from Abraham Mittelmen (note spelling difference) to Bipin Gandhi dated February 12, 1980 dealing with the subject of carbaryl exposure. In this memo dermal absorption rate was estimated by comparing urinary excretion of carbaryl to estimated dermal exposure determined from two different field studies. Estimated dermal absorption ranged from 0.8 to 0.9 percent. These values are 10 times lower than the dermal absorption rate used in Appendix F.

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		It is important to note that these comments, and the estimated exposure values reported in the Mittelman letter identify many of the uncertainties in the risk analysis process. Mittelman used assumptions that differed somewhat from those in Appendix F. However, he estimated that exposure to residents, who may be outside during spraying, would be 0.264 mg/person. Assuming a 70 kg person, this would be a dose of 0.004 mg/kg which is within the range used in the analysis in Appendix F.
	qq	Exposure from drift is discussed for distances of 250 feet, 1/4, 1/2, and 1 mile on page F-19. Only the near range drift (250 feet) was used in the rest of the analysis because the value was by far the highest. Exposure to drift 1000 feet from a treatment site would be considerably less than that at 250 feet.
14		Comments noted.
15		No response necessary.
16		No response necessary.
17	a	See response to comment 11b.
	b	For clarity, the Department has made some editing and modifications. The summary of the FEIS as supplemented does meet the requirements of 40 CFR Section 1502.12.
	c	The Department disagrees. The FEIS as supplemented thoroughly analyses and discusses human health effects of exposure to chemical and biological insecticides that could be used in suppression or eradication projects. In addition, the human health effects of a

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		particular project in any given year are to be evaluated in a site-specific environmental analysis conducted in accordance with NEPA.
	d	The final EIS was available for public comment. See response to comment 11a and 11g.
	e	As previously explained in response to comment 11h. The Department has provided appropriate public comment and review period on this document as required by NEPA.
	f	As was explained in 12c, the only assumption was that 1 pound of any insecticide applied in a similar manner will result in 1 pound of the insecticide being deposited on an acre. This assumption produced the initial residue levels for the specific insecticides. Data for the specific insecticides were then used to evaluate residues on edible plants or meats and how long the residues persist in the environment.
	g	The Department disagrees with your opinion. See the discussion in Appendix F-7 and F-8 for the Department's position on these issues. Also, see response to 12u and comment letter 29 (EPA letter) for further comments on ADIs and safety factors.
	h	As previously noted, see response 11a, 11g, 11h, and 17e. The Department has provided for the appropriate public comment and review period as required by NEPA.

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18	a	Comments were not limited to the Draft Supplement. See responses to comment 11a and 11g. Furthermore, public comment was solicited in 1983, when the Department published its notice of intent to revise the existing 1981 EIS (48FR 46089). Input was also solicited via letter dated October 13, 1983, to cooperating State agencies, environmental groups and the public. Public comment and input on the draft EIS were solicited on the draft EIS in January 1984 (49FR 933 and 49FR 2001) and also on the risk analysis that was added to the Final EIS (49 FR 10963).
	b	The Department disagrees. For comments on the readability of the draft supplement, see comment letters 14, 19b, 19c, and 29. Furthermore, the Department added a writer editor to the writing team. Regarding the adequacy of the summary, the summary to the FEIS as supplemented does meet the requirements of 40 C.F.R. Section 1502.12. See response 17b.
	c	The FEIS as supplemented does discuss the health and environmental effects of using carbaryl (pp. 53-61), the alternatives (pp. 14-24 and pp. 32-76), and public involvement and notification (pp. 77-78). The readability and the summary issues have been previously addressed in response 18b.
	d	For clarity, the Department has made some additions and modifications in the text of the FEIS (pp. 16-17) in order to facilitate a comparative discussion of the chemical insecticides. The FEIS as supplemented discusses alternatives on pages 14-24 and provides a thorough presentation of

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		has been shown to affect population dynamics in limited testing; however, this information has no practical application in the current IPM approach.
	e	The FEIS is intended as an umbrella document addressing the environmental impacts of management activities taken to achieve suppression or eradication (management objectives) of the gypsy moth. Land managers anticipating a need to act against this pest already are aware of their particular management objectives. Where appropriate, these are considered in site-specific environmental analyses. The biological and economic impacts of defoliation and tree mortality can be different for areas with different management objectives and these impacts are most obvious at the local land manager level.
	f	Guidance for conducting site-specific environmental analyses is presented in CEQ Regulations. General information is provided throughout the FEIS and in the NEPA which is referenced throughout the document. Additional technical assistance is provided by APHIS and the Forest Service as needed. The inevitability of the spread of gypsy moth is rather graphically depicted in Table 1, which summarizes annual defoliation by State. In spite of control actions which have taken place since the late 1800's (page 4-6), new States are becoming infested via natural spread. This natural spread into previously uninfested areas from infested areas where no controls are applied occurs at the rate of from 5-15 miles per year.

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20	a	See response to comment 12r and 12w. The actual safety factor suggested in Appendix F for sensitive populations is 10,000 and not 100 as mentioned in the comment.
	b	Cumulative effects are discussed beginning on page F-101 of the FEIS. It was concluded that, because of the additional insecticide residues on store bought foods, it was possible to accumulate a dose that would exceed the ADI for specific insecticides. However residue information provided by FDA indicated that the probability of such accumulation was almost nonexistent.
	c	The discussion on F-101 of the FEIS states, "mitigating measures should be taken so that sensitive individuals, if they can be identified, can avoid direct exposure during application and the eating of food that may contain spray residues." Costs associated with mitigating measures are normally considered part of the project costs and are considered in the decisionmaking process.
	d	The analysis in Appendix F goes far beyond a discussion of just mutagenicity, teratogenicity, or carcinogenicity. No effect levels are given for systemic toxicities for all four insecticides. In fact, the lowest NOELs for acephate, carbaryl, and trichlorfon were for central nervous system toxicity. The lowest NOEL for diflubenzuron was for elevated SHb and MHb, another systemic toxicity. We agree with the commentor, that these are the most sensitive type of toxicities. That was why estimated exposures were compared to the lowest NOEL values as a first measure of risk.

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	e	The actual acreage in Salem, Oregon, receiving multi-year applications of chemical insecticides is 114 acres in two areas. In north Salem, 50 acres were treated with Sevin (2 applications) in 1983. The same acreage was treated again in 1984 with B. t. (3 applications). In south Salem, an area, consisting of 64 acres was treated with Sevin in 1982 (2 applications). The same acreage was again treated with B. t. in 1984 (2 applications).
	f	Synergistic effects were not casually dismissed on F-102. A "worse case" is developed in the last paragraph on this page which assumes a tenfold synergistic factor.
21	a	The majority of dose calculations used in Appendix F were based on urinalysis results from adults. The 70 kg body weight was thought to be appropriate for that situation. The worst case observer dose would increase about 4x if a 15 kg body weight (child) would have been used for the calculation. Dr. Edward Calabrese, who provided a literature review on sensitive populations to USDA, commented on whether age affected the susceptibility of cholinesterase inhibiting insecticides (such as acephate, carbaryl, or trichlorfon). He found that weanling rats are about twice as susceptible as adult rats to parathion, systox, di-syston, guthion, and malathion. A smaller increase in susceptibility occurred with ethion, phosdrin, and carbaryl. However, weanling rats were actually less susceptible to other insecticides including EPN, trithion, and OMPA. Obviously, it

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		is difficult to make generalizations about the sensitivity of children. However, if children are to be considered more sensitive than adults, for equivalent insecticide doses (scaled for different body weights), their responses to exposure would be considered in the discussion of Sensitive Populations. In that case, all no effect levels were reduced by a factor of 100. In other words, children would be 100 times more sensitive than adults.
	b	See response 22e.
	c	See response 20e.
	d	See response to comment 22g.
	e	Additional information regarding heritable mutations has been added on page F-98 and provides an estimated risk of 1×10^{-7} that such mutations could occur from using trichlorfon. This risk to an individual is the same regardless of whether he lives in sparsely or densely populated areas.
	f	The use of B. t. and trapping has shown utility in some but not all situations. This technique is a component of the IPM alternative described in the FEIS. Whether it is appropriate to use B. t. and trapping in combination with, or independent of each other, depends upon site-specific conditions that are addressed in site-specific analyses.
	g	The Department agrees.

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22	a	The report cited, Schulze et al. (dated April 11, 1979), is correct as stated. We have obtained a copy of the undated Schulze et al. report which contains the information enclosed in your letter. As the enclosed paper states, 9 of the 27 residents had positive 1-naphthol readings which ranged from 12.9 to 35.2 ppb. It was concluded that exposure was from secondary sources after spraying, since only one of the nine individuals was on site during application. These values have been included on page F-30 to avoid confusion. It should be noted that these values are well below the value of 247.0 ppb used for this risk analysis.
	b	For purposes of this particular discussion, we feel the cited references are appropriate. The residue levels cited in the reference in the comment letter are within those used in the analysis. To determine a dose for comparison to establish ADIs and NOELs, a dietary component must be calculated. This includes the consumption of animal tissue with insecticide residue. We assume there is no insecticide degradation on vegetation which an animal might consume. This is for calculating a lifetime daily (every day for 25,500 days) dose for humans (a worst case assumption). However, degradation of insecticides in the environment is important in determining cancer rates of the dietary component (food consumed). Deciduous foliage residues were not deemed as appropriate for this purpose.

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	c	EPA has requested new studies on environmental fate for carbaryl. Until these data are collected and evaluated, we will use data from studies reported by Kuhr and Dorough (1976) that show a bioconcentration fact of less than 0.1. This figure (0.1) is consistent with the statement you quote that there may be a potential for accumulation in catfish, crayfish, snails, duckweed, and algae.
	d	The residue levels reported and used in the risk analysis are from published data for evaluations conducted on food crops grown in the United States. We cannot comment on the study mentioned in Organic Gardening because the conditions under which the test took place, the source and purity of the carbaryl, the study design, and protocols are not described. Regarding harvesting schedules, carbaryl will degrade at the same rate independent of the formulation. Therefore, residents who have gardens should follow the same harvesting schedule stated on the Sevin Sprayable label.
	e	The interaction of chemical insecticides is addressed in the synergism/cumulative effect section on page F-101. It was concluded the cumulative doses could possibly exceed the ADIs because of residues on store bought foods or the homeowner use of pesticides. Regarding Dr. Shearer's calculations, an ADI could be exceeded by a 33 lb child touching 2 square inches of soil, foliage, etc., only if the following were assumed: a) residues are 100 percent dislodgable, b) dislodged residues are absorbed 100 percent by humans, and c) the ADI used for

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		could have been used as examples of increased cancer risk, over which individuals exercise no choice.
	h	Chemical insecticides are synthetic chemicals, but not necessarily unnatural substances. Some are human made copies of naturally occurring substances. The body has metabolic mechanisms which normally hydrolyze, detoxify, and metabolize substances which enter the body and actively excrete those substances which it cannot use. These are chemical reactions which will take place whether it is the first time or the 1 millionth time a chemical enters the body.
	i	These concerns are discussed under sensitive populations (F-98) and are addressed by the safety factors included in the establishment of ADI's.
	j	The preparers of the risk assessment were concerned about the interpretation of the toxicology data and whether or not proper risk analysis procedures were followed. This is why the document was submitted to the technical reviewers who are listed with the preparers of the final document. In the environmental analyses developed with cooperating State agencies, public involvement and notification is required. We agree that where appropriate this should include notification of poison control centers and local emergency facilities.
23		No response required.
24		No response required.

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		could have been used as examples of increased cancer risk, over which individuals exercise no choice.
	h	Chemical insecticides are synthetic chemicals, but not necessarily unnatural substances. Some are human made copies of naturally occurring substances. The body has metabolic mechanisms which normally hydrolyze, detoxify, and metabolize substances which enter the body and actively excrete those substances which it cannot use. These are chemical reactions which will take place whether it is the first time or the 1 millionth time a chemical enters the body.
	i	These concerns are discussed under sensitive populations (F-98) and are addressed by the safety factors included in the establishment of ADI's.
	j	The preparers of the risk assessment were concerned about the interpretation of the toxicology data and whether or not proper risk analysis procedures were followed. This is why the document was submitted to the technical reviewers who are listed with the preparers of the final document. In the environmental analyses developed with cooperating State agencies, public involvement and notification is required. We agree that where appropriate this should include notification of poison control centers and local emergency facilities.
23		No response required.
24		No response required.

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25		Every attempt to implement practical and effective mitigating measures are made based upon the results of site-specific environmental analyses conducted in accordance with NEPA for each specific project.
26		No response required.
27	a	An addition was made on page F-2 to reflect this concept. The section on Worst Case Analysis in the body of the FEIS has also been expanded to better define realistic and worst case.
	b	This has been corrected. The teratogenicity NOEL was for rabbits, not dogs.
	c	The paragraph acknowledges that the realistic and worst case doses do not take into account metabolism or excretion of acephate. The Department agrees that the realistic and worst case doses probably greatly overestimate the doses.
	d	See response 27c.
	e	A clarification of cholinesterase inhibition and treatability is added in the FEIS.
	f	The Department does state in the paragraph in question, that the teratogenicity NOELs represent the highest doses tested without an observed effect.
	g	Spills in water result in doses that exceed the ADI by 10 to 18 times only if an individual consumes 2 liters of this water under the assumed conditions stated on pages F-41 to F-42. This is clarified in the paragraph.

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	h	This sentence has been changed to reflect use of EPA validated IBT studies.
	i	Exposure scenarios for goats, rabbits, and fish were developed in order to estimate possible worst case doses for humans who may consume wild game or fish. It is the estimated dose to the human from these sources of food that is added to doses from other exposure pathways and compared to the ADI.
	j	That is correct. It is a deliberate overestimate for project personnel, but one the Department feels is unlikely to be exceeded by any one individual occupationally exposed on a project, accidents excluded.
	k	These represent "worst case" assumptions and are not based on any data set.
	l	Yes. The derivation of ADIs is pointed out on pages F-67 to F-69 and F-87.
	m	Correction made.
	n	Correction made.
28	a	We do not agree with the comment that the "...concept of Integrated Pest Management (IPM) as presented throughout the document is incomplete." The part of the decisionmaking process relating to the need for treatment is covered in site-specific environmental analyses. These decisions are most appropriately made at the local (State) level based on locally developed criteria. Monitoring, when deemed appropriate, is identified in these site-specific analyses.

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	b	In our view, the economic data discussed are sufficient for the purposes of this document. Answers to your questions are contained in the cited references. Detailed discussions of these economic references were not included per 40 C.F.R. Section 1502.21 (Incorporation by reference).
	c	We make no statement and intend no inference in the FEIS that "...pesticides are considered not to be harmful...if acute effects are not observed." The literature cited on page 59 states that the effects were of a temporary nature based on sampling; these effects were not assumed. Additionally, the work cited involves, specifically, the effects of diflubenzuron on the aquatic environment. No assumption is made by the authors, or is intended by the writers of the FEIS regarding the importance of the species diversity versus the overall community numbers. Willcox and Coffey, 1978, as cited, state that population recovery of the more sensitive species occurs within 14 to 28 days in most cases. Individual decisionmakers can judge the relative importances of such factors in arriving at their decisions. We also have made no assumptions regarding sub-acute or chronic effects since, as you imply, there are little or no data in this area.
	d	Predictions of natural spread are based on historical data (5 to 15 miles per year) and are sufficiently accurate to guide regulatory and suppression decisionmaking. A recent APHIS report discussed the development of a computer model to predict spread (USDA 1982b). Host plant quality

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		has been shown to affect population dynamics in limited testing; however, this information has no practical application in the current IPM approach.
	e	The FEIS is intended as an umbrella document addressing the environmental impacts of management activities taken to achieve suppression or eradication (management objectives) of the gypsy moth. Land managers anticipating a need to act against this pest already are aware of their particular management objectives. Where appropriate, these are considered in site-specific environmental analyses. The biological and economic impacts of defoliation and tree mortality can be different for areas with different management objectives and these impacts are most obvious at the local land manager level.
	f	Guidance for conducting site-specific environmental analyses is presented in CEQ Regulations. General information is provided throughout the FEIS and in the NEPA which is referenced throughout the document. Additional technical assistance is provided by APHIS and the Forest Service as needed. The inevitability of the spread of gypsy moth is rather graphically depicted in Table 1, which summarizes annual defoliation by State. In spite of control actions which have taken place since the late 1800's (page 4-6), new States are becoming infested via natural spread. This natural spread into previously uninfested areas from infested areas where no controls are applied occurs at the rate of from 5-15 miles per year.

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		Criteria used in regulating new areas are based on the finding of more than one life stage of gypsy moth or patterns of male moth trap catches from delineating surveys in non-regulated areas which strongly suggest that reproduction is taking place. Because of the nebulous nature of the term "leading edge", no relationship between it and non-regulated areas has been defined. The monitoring and control goals of the Forest Service in infested areas are stated on page 12, paragraph 1, and page 12, paragraph 4 of the FEIS. Monitoring, or more appropriately survey and eradication of isolated infestations are APHIS goals and are discussed on page 12, paragraph 2, and page 12, paragraph 3 of the FEIS.
	g	The USDA Forest Service cooperates in the funding of gypsy moth suppression projects based on requests from the States. These requests are based upon criteria developed in site-specific environmental analyses conducted in accordance with NEPA.
29	a	No response necessary.
	b	The factor for <u>in vitro</u> yield has been removed (see response to letter 13n). Only the <u>in vivo</u> yields are needed to calculate N-nitrosocarbaryl from carbaryl concentrations. The reference for <u>in vivo</u> yield (Rickard and Dorough 1979) has been added to F-74. The explanation of how cancer potency (β) was derived is on pages F-14 to F-17. As noted on F-16, the method of calculation was a variation of that described by Crouch and Wilson (1979).

Letter No.	Comments	Response
	c	The requested addition has been made to page F-15. The studies required under data call-in for trichlorfon (because of the registration standard) were discussed on page F-9. The oncogenicity study and studies needed to re-establish the ADI were just two of many studies identified on page F-9.
	d	This comment also emphasizes the fact that there are two types of data gaps: those that relate to FIFRA and the registration process and those that refer to NEPA. A prudent responsible official can make reasoned decisions about the risks of various alternatives even though that decision may be based in part on data that has been judged to be inadequate for registration purposes. In such cases, the uncertainties in the data need to be evaluated or discussed, as was the case in the FEIS.
	e	No response necessary.
	f	The qualification about the trichlorfon NOEL of 1.25 mg/kg/day has been added to F-67 to F-68.
	g	Because of this comment and those by NCAP (letter 12), a full cancer risk analysis has been added to the FEIS. This analysis was based on information provided by Chevron Chemical Company.
	h	The first reference in Table 1, which was subchronic feeding study in rats conducted by IBT, has been removed from the Table. Since cholinesterase inhibition was measured at the same dose level (5 ppm) in the 28-month ongenicity study in rats, this value was used in the analysis as the NOEL for cholinesterase inhibition.

Letter No.	Comments	Response
	i	All requested corrections have been made based on the new lowest NOEL value of 1.1 mg/kg/day and the safety factor of 100.
	j	Corrections to F-67 and F-87 have been made. ADI is now referred to as "a concept" instead of a "standard".
	k	All references to consumption of "contaminated" food or water have been replaced with references to food or water that may "contain pesticide residues."
30		No response required.
31		No response required.
32		No response required.

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